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ENDOCRINE TREATMENT OF METASTATIC BREAST CANCER

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New York City

A variety of alterations in the endocrine environment will induce temporary cessation of growth and regression of metastatic mammary cancer in some women. Thus, such different procedures as oophorectomy, adrenalectomy and hypophysectomy, or the administration of estrogen, androgen and cortisone may produce objective remissions in patients with breast cancer. It is the purpose of this paper to review the results of these several modalities of endocrine treatment, to consider the mechanisms by which alterations in endocrine environment induce remissions, and to outline the sequence of therapy which is most likely to yield optimum therapeutic results.

OOPHORECTOMY

Beatson[3], in 1896, was the first to demonstrate that oophorectomy could induce regression of metastatic mammary cancer in women. This observation has been amply confirmed by many investigators. Oophorectomy has not come into general use in the therapy of metastatic breast cancer perhaps because of the fact that many women fail to benefit from this procedure.

We recently have reviewed the results of oophorectomy in 75 premenopausal women with metastatic breast cancer.[17] Forty-four per cent of these women obtained objective remissions of their disease. The average duration of the remissions was 9 months, with a median duration of 6.5 months. Metastases to nearly every organ have been observed to regress following oophorectomy in this group of patients. Oophorectomy produced worthwhile palliation in those patients who had a favorable response.

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* American Cancer Society Scholar, † McGlaughlin Fellow, ‡ Damon Runyon Research Fellow, § U. S. Public Health Service Fellow.

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TABLE I

Results of endocrine treatment for metastatic breast cancer

Treatment	Status	Number of Cases	Objective Remissions %	Duration (months)	
				Mean	Median
Oophorectomy (17)	Premenopausal	75	44	9	6.5
	Postmenopausal	21	10	—	—
Adrenalectomy (17)	(All previously Oophorectomized)	38	45	8.5	7
Combined Oophorectomy and Adrenalectomy (25)	Postmenopausal	25	64	(7+)	(7+)
*Hypophysectomy (20)	All ages	30	53	—	—
Androgen (21) (Testosterone Propionate 100 mg. 3 times a week)	Premenopausal	54	15	10	4
	Postmenopausal	113	20	8	8
Estrogen (21) (Stilbesterol 15 mg./day or Estinyl 3 mg./day)	Postmenopausal	76	32	13.5	8
Cortisone (24)					
200–400 mg./day	All ages	39	31	3	3
50–75 mg./day	All ages	14	14	2	—

* The majority of these patients had undergone previous endocrine treatment prior to hypophysectomy. Ten patients had oophorectomy and adrenalectomy, 4 of whom obtained further remissions from hypophysectomy.

No satisfactory means for selecting patients for oophorectomy has been found. Neither the histologic picture of the cancer nor any clinical manifestations of the disease has been helpful in predicting a favorable response.

Pearson and associates[18] have demonstrated that there are two types of breast cancer in women. Approximately 50 per cent of patients have estrogen-dependent mammary cancer, whereas the rest have nonestrogen dependent tumors. Oophorectomy removes a major source of estrogen and induces temporary objective improvement in patients with estrogen-dependent breast cancer. Castration fails to alter the growth of nonestrogen dependent cancer. These observations provide a physiologic basis for the treatment of mammary cancer in women.

Oophorectomy is the initial treatment of choice in premenopausal women with metastatic breast cancer, since this procedure yields the highest percentage of favorable results. In postmenopausal women, oophorectomy occasionally will produce improvement (table I). Since less than 10 per cent of patients respond, combined oophorectomy and adrenalectomy is the preferred treatment in the postmenopausal patient (*vide infra*).

ADRENALECTOMY

Huggins and Bergenstal[10] were the first to report remissions in women with breast cancer following adrenalectomy. West and co-workers[22] first demonstrated conclusively that removal of the adrenal glands in women with breast cancer who had previously had oophorectomy could produce regression of the tumors. Of 38 women with metastatic breast cancer, all of whom had previously had

oophorectomy, bilateral adrenalectomy produced objective remissions in 45 per cent of the cases.[17] The average duration of the remissions was 8.5 months, with a median of 7 months. Worthwhile palliation was obtained in most of the patients who obtained remissions.

The mechanism by which adrenalectomy induces remissions in women with breast cancer is considered to be the removal of another source of endogenous estrogen. Thus, only patients with estrogen-dependent mammary cancer should obtain benefit from adrenalectomy. This has been our experience so far. Of 17 patients who obtained objective remissions from oophorectomy, 9 obtained second remissions following adrenalectomy; whereas, 6 patients who failed to respond to oophorectomy also failed to respond to adrenalectomy.

Combined oophorectomy and bilateral adrenalectomy in one stage has been evaluated in 25 postmenopausal women* with metastatic breast cancer.[25] Sixteen of these 25 patients obtained objective remissions. The average duration of the remissions was greater than 7 months (8 of the patients still are in remission). These observations indicate that approximately 50 per cent of women of all ages have estrogen-dependent mammary cancer and will respond favorably to removal of endogenous sources of estrogen.

The histologic picture of the tumor and the clinical features of the disease have been of no help in selecting patients for adrenalectomy. In premenopausal patients, a favorable response to oophorectomy indicates that there is better than a 50 per cent chance of further improvement being obtained from adrenalectomy. Failure of the premenopausal patient to respond to oophorectomy usually means failure to respond to adrenalectomy. Thus, knowledge of the response of the premenopausal patient to surgical castration provides the best means of selecting patients for adrenalectomy. This information is, of course, of no value in the postmenopausal patient. It also has been our experience that patients who obtain an objective remission from androgen therapy are likely to obtain further remissions from adrenalectomy or from combined oophorectomy and adrenalectomy. The converse of this is not true, however, because a number of patients who failed to benefit from androgen therapy have obtained objective benefit from adrenalectomy or combined oophorectomy and adrenalectomy.

In premenopausal patients who have responded to oophorectomy and then relapsed, adrenalectomy is the treatment of choice, since this procedure offers the greatest percentage of favorable results. In postmenopausal women, combined oophorectomy and adrenalectomy is the recommended initial therapy.

HYPOPHYSECTOMY

Luft and Olivecrona[13] were the first to report a remission from hypophysectomy in a patient with advanced breast cancer. Since the ovaries and adrenal glands had not been removed from their patient, it could not be determined whether the remission resulted from suppression of ovarian and adrenal func-

* Of these 25 patients, 15 had spontaneous menopause and 10 had artificially induced menopause. 12 of the 15 spontaneous menopause cases and 4 of the 10 artificial menopause cases had remissions,

tion consequent to hypophysectomy, or whether removal of pituitary hormones *per se* was responsible for the improvement. Pearson and associates[19] were the first to demonstrate objective remissions in women with metastatic breast cancer who had previously had oophorectomy and adrenalectomy. Following hypophysectomy, administration of beef pituitary growth hormone to 1 patient apparently accelerated the growth of the tumor, which subsided when the growth hormone was withdrawn. This observation suggests that some breast cancers may be growth hormone dependent.

Hypophysectomy produces profound suppression of the function of the ovaries and adrenal glands, comparable to the surgical ablation of these endocrine organs.[23] Thus, it would appear that hypophysectomy can produce the beneficial effects to be obtained from castration and adrenalectomy and, in addition, produce any further benefit that may be obtained from removal of pituitary hormones.

Pearson, Ray and co-workers[20] have obtained objective remissions following hypophysectomy in a selected series of 16 of 30 women with advanced breast cancer. Of 10 patients who previously had undergone surgical castration and adrenalectomy, 4 obtained further remissions from hypophysectomy.

Further experience with hypophysectomy in the treatment of advanced breast cancer is needed before a final appraisal can be made, but these preliminary observations suggest that hypophysectomy may offer the optimum endocrine therapy for these patients.

ANDROGEN

Loeser[12] and Ulrich[21] were the first to report objective improvement in patients with metastatic breast cancer from testosterone administration. It has been well established by many investigators that androgen therapy will induce objective improvement in about 23 per cent of women with breast cancer in all age groups.[2] The average duration of the remissions is about 7.5 months, with a median of about 4.5 months.[4]

The mechanism by which androgen induces remissions in patients with breast cancer is not known. It has been considered that androgen may neutralize the effect of estrogen, although no direct evidence has been obtained to support this hypothesis. Androgen frequently produces remissions in patients with estrogen-dependent breast cancer. We have never obtained a definite remission with the use of androgen in a patient with nonestrogen dependent mammary cancer, although more experience is needed in this group of patients. If this is the mechanism of androgen action, it does not appear to be an efficient neutralizing agent, since approximately 50 per cent of women have estrogen-dependent mammary cancer and androgen is effective in only 20 per cent of cases.

Farrow and Woodard[6] and, more recently, Myers and co-authors[14] have demonstrated that androgen may accelerate the growth of mammary cancer in some patients. Estrogen also accelerated the growth of the tumor in the same patients. Nathanson and associates[15] have presented evidence which suggests that androgen may be metabolized in the body into estrogenic substances. If it can be con-

clusively demonstrated that androgen can be converted into estrogen in some patients, this will offer a plausible explanation of the way in which androgen produces exacerbation of tumor growth in some patients, and why androgen fails to benefit some patients with estrogen-dependent mammary cancer.

Since androgen therapy carries the potential of exacerbating the growth of breast cancer, and since the percentage of remissions obtained with androgen is less than those obtained with oophorectomy and adrenalectomy, it would seem logical to reserve androgen therapy until the disease has relapsed after oophorectomy and adrenalectomy. Androgen therapy also is indicated in those patients who cannot tolerate or who refuse ablative therapy.

If the mechanism of androgen action is estrogen neutralization, it is of interest that androgen therapy has been effective in some patients after oophorectomy and adrenalectomy. This would suggest that oophorectomy and adrenalectomy have not removed all of the endogenous sources of estrogen. The presence of accessory adrenal glands in many patients may represent the site of such hormone production[5, 7]. It will be of considerable interest to determine whether androgen therapy will be effective in patients whose disease relapses after hypophysectomy.

ESTROGEN

Haddow and associates[8] were the first to report regression of metastatic breast cancer with the use of estrogen. Nathanson[16] and Herrman, Adair and Woodard[9] confirmed these observations and found that estrogen was more likely to be effective in the older postmenopausal patient. Escher and co-workers[4] obtained objective remissions in 32 per cent of postmenopausal women with the use of estrogen. The average duration of the remissions was 13.5 months, with a median of 8 months.

Estrogen appeared to accelerate the course of the disease in some patients. Great caution must be exercised with the use of estrogen. If the disease continues to progress, the hormone should be promptly discontinued. It has been recommended that estrogen should be restricted in its use to the older postmenopausal patient.[9]

The mechanism by which estrogen induces remissions in women with breast cancer has not been established. It seems possible that estrogen may induce beneficial effects by suppression of pituitary function. Estrogen will decrease the excretion of follicle-stimulating hormone in the urine of postmenopausal women. Albright[1] and others[11] have induced remissions in patients with acromegaly by the use of estrogens. Thus, if breast cancer in some women is dependent upon certain pituitary hormones for growth, the suppression of pituitary hormone secretion by estrogen would offer an explanation for the beneficial effects of estrogen in these women. More definitive observations are required before such an explanation of estrogen action can be established.

It has been well established that estrogen can accelerate the growth of breast cancer in some women.[18] Since approximately 50 per cent of women of all age groups appear to have estrogen-dependent breast cancer, estrogen is a potential hazard in this group of patients. Some breast cancers may have a dual dependence

upon hormones, e.g., estrogen and growth hormone. Thus, it is possible that estrogen might have dual effects on the growth of the cancer; one to stimulate and the other to suppress, of which either effect might predominate. These theoretical considerations offer a possible explanation for the paradoxical effects of estrogen in patients with breast cancer.

Since estrogen potentially is capable of producing harmful effects in about 50 per cent of all women with breast cancer, it would seem wise to restrict its therapeutic use to those patients who fail to respond to castration or to castration and adrenalectomy. Estrogen therapy should be tried cautiously when ablative procedures cannot be done.

CORTISONE

Pearson and associates[18] were the first to report objective improvement in women with breast cancer with the use of large doses of cortisone (200–400 mg./day). West and associates[24] obtained objective remissions in 31 per cent of their cases with high-dosage cortisone. These workers also treated 14 patients with small doses of cortisone (50–75 mg./day) and obtained objective remissions in 2 of these patients. The average duration of the remissions was 3 months.

Since cortisone is known to suppress the function of the adrenal cortex, this would appear to offer a plausible explanation for the beneficial effects of cortisone, particularly in those patients who received the smaller dosage of cortisone. Since only 2 of 14 patients responded to this dose of cortisone, it would appear that cortisone is not as effective as adrenalectomy. Further observations with the use of small doses of cortisone in patients with estrogen-dependent mammary cancer, whose disease is in relapse after oophorectomy, would be desirable. Large doses of cortisone have induced objective remissions in patients who had previously had oophorectomy and adrenalectomy. This observation indicates that large doses of cortisone suppress tumor growth by some mechanism other than suppression of adrenal function. The mechanism by which large doses of cortisone inhibit the growth of some breast cancers is not known.

Since the remissions induced by cortisone usually are of short duration, it seems best to reserve this therapy for patients who fail to respond to the other modalities of endocrine therapy or who have relapsed after such therapy. Cortisone therapy also is useful in critically ill patients. If a remission can be induced with cortisone, the patient then may be suitable for other forms of endocrine therapy.

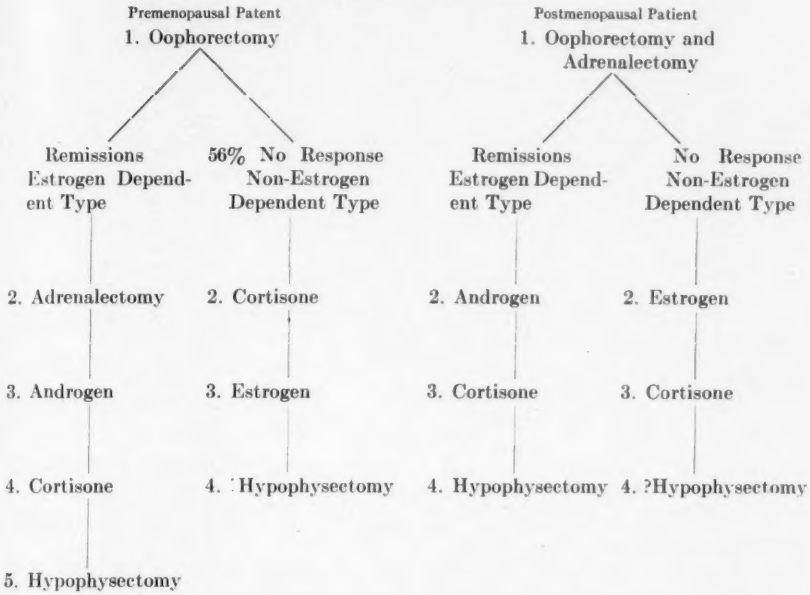
DISCUSSION

Table I summarizes the results of the various forms of endocrine therapy for metastatic breast cancer obtained at Memorial Center. In table II the sequence of endocrine treatment which, at present, offers the optimum therapeutic results is presented. This recommended sequence of therapy will undoubtedly be modified as more experience is accumulated.

The possible mechanisms by which alterations in endocrine environment af-

TABLE II

Recommended sequence of therapy for metastatic breast cancer



fect the growth of breast cancer have been presented. The present evidence suggests that the growth of mammary cancer is, in many cases, dependent upon circulating hormones which are secreted from the ovaries, adrenals and pituitary gland. The removal or lowering the titre of these hormones results in temporary cessation of growth and regression of the cancer. This concept provides a rational, physiologic basis for treatment.

All forms of endocrine therapy, studied so far, produce only temporary palliative effects. Whether this means that breast tumors are only partially dependent upon hormones for their growth, or whether the essential hormones have not been completely eliminated, has not been definitely determined. Since hypophysectomy offers the greatest possibility of eliminating hormones upon which tumor growth may be dependent, further experience with this procedure will be of great interest. It already is apparent, however, that the growth of certain breast cancers is virtually independent of the hormonal environment.

Whether endocrine therapy significantly prolongs life, in those patients with breast cancer who obtain a favorable response, has not been rigidly established on the basis of statistics. On the basis of individual experience, there appears to be no question that useful survival is prolonged in most patients who obtain a favorable response. Carefully planned endocrine therapy constitutes worthwhile therapy for many patients with metastatic breast cancer.

SUMMARY

The results of oophorectomy, adrenalectomy, hypophysectomy and the administration of estrogen, androgen and cortisone for the treatment of metastatic mammary cancer in women are reviewed.

The sequence of endocrine therapy which offers the optimum therapeutic results is presented.

The mechanisms by which alterations in the hormonal environment affect the growth of mammary cancer are discussed and a physiologic concept for treatment is presented.

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VENTRICULAR FIBRILLATION IN THE HYPOTHERMIC STATE

II. THE EFFECT OF SINO-AURICULAR NODE BLOCKADE IN PREVENTING VENTRICULAR FIBRILLATION AT LOW DEGREES OF BODY TEMPERATURE

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Ventricular fibrillation constitutes the greatest danger of acutely induced general hypothermia. From experiments recorded in the literature it seems that the hazard of ventricular fibrillation increases as the body temperature is reduced. It is generally thought that minimal cardiac trauma may produce ventricular fibrillation at temperatures below 24 C. and that fibrillation is likely to supervene without direct cardiac stimuli at temperatures of from 20 to 21 C.

We have demonstrated previously that blockade of the sino-auricular node with procaine hydrochloride is highly effective in preventing ventricular fibrillation in dogs subjected to different types of cardiac stimulation at body temperatures ranging from 24 to 28 C.[4] The present report deals with similar experiments made upon dogs cooled to a body temperature of from 19 to 22.5 C.

MATERIALS AND METHODS

Healthy mongrel dogs were used. Anesthesia was induced by the intravenous injection of from 3 to 4 cc. of 5 per cent thiopental sodium. An endotracheal tube was passed and oxygen was administered for the remainder of the experiment by means of an intermittent positive pressure machine with full inflation of the lungs from 35 to 45 times per minute. Hypothermia was induced by immersion of the animal in a bath of water and cracked ice. When shivering occurred it was controlled by temporary endotracheal administration of a mixture of ether and oxygen. When a temperature of from 22 to 24 C. was reached the animal was removed from the bath and a right thoracotomy was done under aseptic conditions through the fourth intercostal space.

The venae cavae were dissected free and the azygos vein was divided between ligatures. The pericardium was opened widely anterior to the course of the right phrenic nerve. The venae cavae were occluded with Pott's ductus clamps. After 3 minutes of caval occlusion two traction sutures were placed in the right ventricular wall and a wide ventriculotomy was made. The ventricular septum was grasped with forceps and 2 silk sutures were placed in it and tied snugly. The right ventricle then was closed with a continuous suture of silk. Before the last stitch was drawn tight a little blood was allowed to pass into the right ventricle by temporarily releasing the superior vena caval clamp in order to expel any air which might be remaining within the ventricular chamber. The vena caval oc-

From the Department of Surgery, Indiana University School of Medicine, Indianapolis, Indiana. Aided by grants from the James Whitcomb Riley Memorial Association, the Indiana Heart Foundation and by a contract between the Office of Naval Research, the United States Navy and Indiana University.

clusion was continued for a minimum of 10 minutes. The longest period of caval occlusion was 15 minutes. The superior vena caval clamp was released first and the clamp from the inferior vena cava was removed only after cardiac contractions were improving in quality. The period between the release of the two clamps was generally 2 or 3 minutes. Following the removal of the clamp from the superior vena cava the dog was placed in slight Trendelenberg position.

In half of the 32 experiments performed the sino-auricular node was blocked by the injection into the right atrial wall of one per cent procaine hydrochloride. In treated and control animals electrocardiographic tracings were made from time to time during the course of the experiment.

Whenever ventricular fibrillation occurred during the ventriculotomy the incision in the ventricle was closed rapidly and clamps from both venae cavae were removed. In those instances in which no ventricular fibrillation occurred the strength of the cardiac contractions increased slowly after restoration of circulation, but the strength of the ventricular contractions was not as great as is commonly observed at higher body temperatures. In a number of animals it was necessary to carry out cardiac massage for a short period of time following release of the occluding caval clamps in order to secure adequate blood flow and to promote better ventricular contractions. Occasionally, a period of from 10 to 15 minutes passed following restoration of venous inflow into the heart before the cardiac beat appeared entirely satisfactory. Once the cardiac rate, rhythm, and strength of contractions appeared to be satisfactory the pericardium was loosely closed with interrupted silk sutures and the chest wound was repaired in the usual manner.

The animals then were rewarmed by placing them in a water bath maintained at approximately 45 C. They were kept in the bath until the rectal temperatures reached 35 or 36 C. A number of dogs were killed immediately following rewarming and resumption of spontaneous respiration. Others were observed for periods of from 1 to 10 days following the experiments.

RESULTS

The dogs weighed from 7 to 11 kilograms and the time required for reduction of their rectal temperatures to a level of from 22 to 24 C. varied between 45 and 70 minutes. By the time the cardiotomy was performed the temperature usually had dropped another 3 or 4 degrees.

It was a striking observation that the myocardium remained pink for a longer period of time following vena caval occlusion than is the case under similar circumstances at higher body temperatures. In contrast, however, at these low body temperatures the ventricular contractions seemed to be weaker and the rate definitely slower. Often the rate fell to from 5 to 10 per minute before the vena caval clamps were released. Similarly when circulation was restored by removal of the vena caval occlusion a rather long time elapsed before satisfactory contractions were reestablished and frequently cardiac massage had to be employed from time to time in order to prevent threatened cardiac standstill. It also was an interesting observation that most of the animals did not have as

fully dilated pupils during absence of venous inflow into the heart as is commonly observed under similar circumstances in animals with somewhat higher rectal temperatures. Following restitution of circulation the pupils remained dilated for a longer period of time, however, and usually were still half dilated by the time the temperature had been brought up to a level of from 29 to 30 C. Spontaneous respirations were not resumed until a temperature of 34 C. was reached in spite of the fact that anesthesia was kept at a very light level.

Following procaine blockade of the sino-auricular node the heart rate slowed very precipitously. Sometimes, indeed, a momentary cardiac standstill ensued and lasted for a period of 3 to 4 seconds after which the heart would resume a normal rhythm but at a very slow rate. The P waves disappeared from the electrocardiographic tracing in almost all instances or great reduction in the voltage of the P waves took place (figs. 1 and 2).

During all the intracardiac procedures and during the total period of vena caval occlusion the cardiac rhythm continued to be regular both in the treated

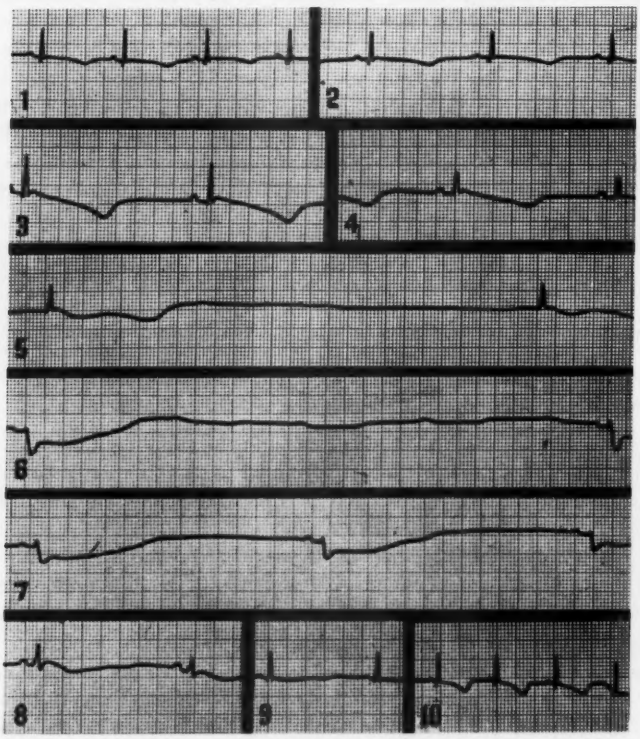

FIG. 1. Electrocardiographic tracings (Lead 2) in dog number 26. 1. At 34 C. 2. At 27 C. 3. At 24 C. 4. At 21 C. before injection of procaine. 5. After injection. 6. Upon release of superior vena caval clamp. 7. Upon release of inferior vena caval clamp. 8. After closure of pericardium. 9. At 28 C. 10. At 36 C.

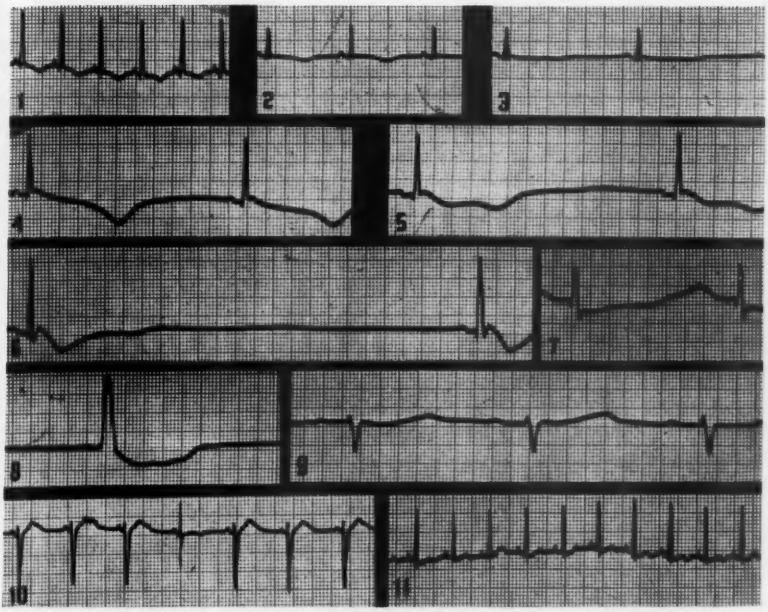

FIG. 2. Electrocardiographic tracings (Lead 2) in dog number 30. 1. At 37 C. 2. At 30 C. 3. At 25 C. 4. At 20 C. before injecting procaine. 5. And 6. Immediately, and 1 minute after injection. 7. After release of both vena caval clamps. 8. Very slow rate—5 per minute. 9. After administration of intravenous dilute epinephrine solution. 10. At 36 C. 11. Two days later.

animals and in those in which sino-auricular node blockade was not carried out but in which fibrillation did not occur. Except for those in which fibrillation took place the hearts of the untreated animals appeared to be as regular in rhythm and as free of irritability as those treated by sino-auricular blockade. Most of the animals demonstrated low voltage and inversion of the ventricular complex after ventriculotomy. These alterations usually disappeared during the rewarming period and the tracings usually were normal by the time body temperatures of from 29 to 30 C. were reached.

Most treated and untreated animals required special care during the period of rewarming. It was found that the endotracheal tube could not be safely removed even after spontaneous respirations had been resumed and body temperature raised to relatively normal levels because of the recurrence from time to time of sudden cessation of respiratory movements necessitating artificial respiration. Furthermore, in both groups of animals there was noted a tendency to the occurrence of cardiac standstill, not only immediately after the release of caval occlusion but also during the rewarming period up to a point at which the body temperature had reached 30 degrees or greater. When standstill appeared imminent it was found that the intravenous administration of a few cc. of dilute solution of epinephrine (1 ampule of 1 to 1000 in 500 cc. of normal saline) usually

brought about a dramatic improvement in the cardiac rate, strength of contractions, and arterial blood pressure. Administration of the weak epinephrine solution was discontinued as soon as such a good response was noted.

Nine of the 16 control animals developed ventricular fibrillation, an incidence of 56.2 per cent (table 1). Cardiac standstill developed in 1. Six, or 37.5 per cent, survived the procedure and rewarming.

TABLE 1

Effect of sino-auricular node blockade on incidence of ventricular fibrillation in hypothermic dogs subjected to right ventriculotomy and placement of sutures in ventricular septum

No. of Dog	Temperature in degrees centigrade	Duration of caval occlusion in minutes	Result
		TREATED ANIMALS	
1	21	10	No fibrillation
5	22	10	No fibrillation
6	20	10	No fibrillation
8	21	10	No fibrillation
12	22.5	10	No fibrillation
13	22	10	No fibrillation
14	21.8	10	No fibrillation
16	19.8	10	No fibrillation
17	19	10	No fibrillation
18	20	10	No fibrillation
21	20	10	No fibrillation
23	19	15	Fibrillation during massage
25	21.5	10	No fibrillation
26	21	10	No fibrillation
29	19	10	Cardiac arrest during rewarming
30	20	10	No fibrillation
		UNTREATED CONTROL ANIMALS	
2	22	10	No fibrillation
3	21.5	10	No fibrillation
4	22	8	Fibrillation
7	21	5.5	Fibrillation
9	22.5	3	Fibrillation
10	22	10	Fibrillation upon removal of superior vena caval clamp
11	22	8	Fibrillation
15	21	10	Standstill
19	20	10	No fibrillation
20	20	3	Fibrillation
24	19.8	10	Fibrillation during rewarming
27	20	10	No fibrillation
28	19.5	10	No fibrillation
31	20	10	No fibrillation
32	20	7	Fibrillation
33	20	8	Fibrillation

In the treated group of animals ventricular fibrillation occurred in only a single instance. This complication took place in the only animal in which vena caval occlusion was maintained for a period of 15 minutes and developed not during the ventriculotomy but during a period of cardiac massage following release of vena caval occlusion. In 1 additional animal cardiac standstill took place during the rewarming period. In this instance intravenous administration of a weak epinephrine solution was not utilized and from other experiences one would suppose that such treatment probably would have been effective.

DISCUSSION

The reported experiments demonstrate that procaine blockade of the sino-auricular node is very effective in preventing ventricular fibrillation during vena caval occlusion and right ventriculotomy in normal dogs cooled to body temperatures in the range of from 19 to 22.5 C. Indeed, of 16 animals thus treated ventricular fibrillation occurred in only 1 and in this exceptional case developed during cardiac massage carried out because of weak contractions which followed a 15 minute period of vena caval occlusion. In no other animal was the period of caval occlusion so long. In contrast 9 of the 16 untreated control animals developed ventricular fibrillation.

It has been emphasized by a number of investigators that ventricular fibrillation is more likely to occur below temperatures of 25 to 26 C.[2-5] In our own studies ventricular fibrillation developing as the result of right ventriculotomy and prolonged inflow occlusion did not occur as frequently at temperatures in the neighborhood of 20 C. as was true at body temperatures in the neighborhood of 26 to 28 C. In the present experiments performed at the lower body temperatures the incidence of ventricular fibrillation was 56 per cent. In previous experiments carried out at higher temperatures the incidence of ventricular fibrillation under the same circumstances was 100 per cent. In animals cooled to the lower temperatures, however, there were noted certain difficulties which were not encountered in experiments at higher body temperatures. There was a greater tendency for the occurrence of cardiac standstill during the period immediately following release of caval occlusion and during the rewarming period. This proneness to the development of cardiac standstill apparently could be effectively combatted by the intravenous administration of minute quantities of epinephrine solution. There also was noted an unexplained tendency in some animals for spontaneous cessation of respirations during the period following body rewarming. When such difficulties occurred and respirations were momentarily maintained by artificial insufflation of the lungs, spontaneous respirations usually were resumed and recovery ensued. This complication generally was encountered during periods in which the body temperature was observed to have fallen precipitously and under such circumstances it was necessary again to rewarm the animal.

SUMMARY

The demonstration that sino-auricular node blockade apparently is effective in preventing ventricular fibrillation during venous inflow occlusion and right

ventriculotomy at relatively low body temperatures may be of some importance with regard to efforts to increase the safe period of vena caval occlusion and ventriculotomy in the hypothermic state.

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INTESTINAL OBSTRUCTION. II. ANALYSIS OF 1287 ADMISSIONS OVER A TEN YEAR PERIOD*

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INTRODUCTORY REMARKS

Intestinal obstruction still is a leading cause of surgical morbidity and mortality in most medical centers. In an effort to reassess our treatment for this condition, we have examined the results of 1287 admissions over a 10 year period at the John Gaston Hospital, the charity hospital of Memphis and Shelby County and the teaching hospital of the University of Tennessee. Patients treated at this hospital almost entirely come from an indigent segment of the population that even yet is not aware of the importance of early treatment for this and other pathologic conditions. These patients often are admitted to the hospital in states of extreme electrolyte and nutritional depletion, and for this reason the mortality rates are somewhat higher than those encountered in private practice.

The results of the management of intestinal obstruction in this hospital from 1923 to 1932 were reported by Tendler.[2] He found the over-all mortality rate to be 50.9 per cent, which at that time compared favorably with that of other hospitals of this type. However, due to newer concepts of fluid, electrolyte, and colloid therapy, intubation and suction, chemotherapeutic agents, and an ever increased realization that prompt diagnosis and treatment are essential to improved therapy, this mortality rate was steadily reduced in the following years. In a later survey of the incidence and the mortality rate from intestinal obstruction in this institution, covering the years 1941 to 1946, there was an over-all mortality rate of 27.4 per cent.[2] Thus, in approximately 10 years the mortality rate was reduced by 50 per cent. This reduction resulted more from improved methods of treatment than from an increased patient awareness of the need for earlier medical aid; even at the present time patients are regularly admitted to the hospital in a state of severe depletion from far advanced complete obstruction.

MATERIAL AND RESULTS

The material analyzed included 1287 admissions—1131 different patients—who were treated for intestinal obstruction from all causes over a 10 year period (Jan. 1, 1944, through Dec. 31, 1953). The diagnosis was established on the basis of physical and roentgenologic examination and at operation. All patients in whom the diagnosis of intestinal obstruction was established were included in the survey. However, since many were moribund upon arrival, special mention is made of that group of patients who were so severely ill that they died prior to

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receiving actual therapy. Not included in the study were the following: (a) obstruction of the pylorus or first portion of the duodenum by chronic ulcer or malignancy; and (b) adynamic (paralytic) ileus.

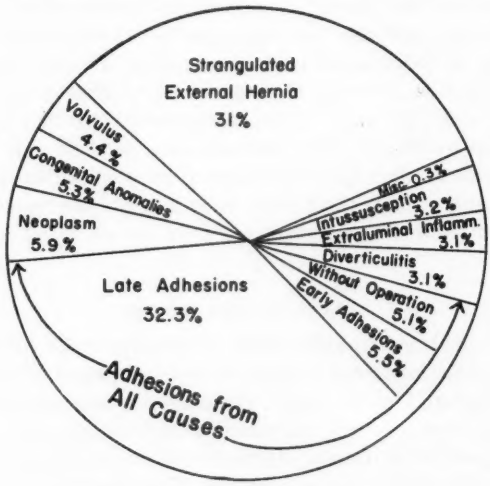
Of the 1287 admissions, 937 patients were treated by operation and 350 were treated conservatively.

CAUSES OF OBSTRUCTION

In the present series, there were 1128 instances of small bowel obstruction and 159 instances of large bowel obstruction (fig. 1 and table I). As in most large surveys, the majority of cases were due to adhesions (42.9 per cent), hernias (31.0 per cent), and neoplasms (5.9 per cent), these three comprising 79.8 per cent of the total.

Adhesions. In this category were 554 cases or 42.9 per cent of the total, and the majority of these were instances of obstruction of the small bowel (549). Thus, obstruction of the large bowel occurred as a result of adhesions in only 5 patients. Of the 554 cases of obstruction due to adhesions, 88.5 per cent followed operation, 14.2 per cent of these occurring less than one month after operation and 85.8 per cent occurring thereafter. The remaining 11.5 per cent were due to inflammatory disease or to other causes.

Hernias. By far the second most frequent cause of obstruction were the various types of hernias (table II). This group comprised 32.6 per cent of the total (419 cases). Indirect hernia constituted the majority (67 per cent) of these, with the right indirect hernia alone accounting for 47 per cent of the obstructions due to



CAUSES OF INTESTINAL OBSTRUCTION IN 1,287 ADMISSIONS

FIG. 1

TABLE I

Causes of obstruction

	Small Bowel	Large Bowel
Adhesions	549	5
Hernia	459	0
Neoplasm	15	62
Congenital anomalies	51	25
Volvulus	12	45
Intussusception	40	2
Pelvic abscess	15	4
Appendiceal abscess	12	0
Fecal impaction	0	8
Crohn's disease	5	0
Diverticulitis	0	5
Mesenteric thrombosis	4	0
L.V. stricture	0	2
Gallstone ileus	2	0
Miscellaneous	4	1
Totals	1128	159

TABLE II

Hernia

Indirect, right	218
left	90
Umbilical	41
Femoral, right	15
left	12
Internal	16
Ventral	8
Incisional	8
Epigastric	5
Diaphragmatic	3
Direct, right	1
left	1
Bilateral, indirect	1

hernias. Internal hernias were exceedingly rare, and there were but three hernias of the diaphragmatic type.

The marked infrequency with which direct hernias caused obstruction is clearly demonstrated in table II. Only two instances were due to inguinal hernias of the direct type. These represented only 0.14 per cent of the total cases and 0.43 per cent of the obstructions caused by hernia.

Neoplasms. This group of lesions represented the third most important category, constituting 5.9 per cent of the total number of cases. Malignant processes were by far the most important causes of neoplastic obstructions in both the large and small bowel. Of the 62 instances of large bowel obstruction caused by

TABLE III

Neoplasms

A. *Large Bowel*	
Adenocarcinoma—Cecum	9
Adenocarcinoma—Transverse colon	3
Adenocarcinoma—Descending colon	3
Adenocarcinoma—Sigmoid	10
Adenocarcinoma—Splenic flexure	15
Adenocarcinoma—Hepatic flexure	3
Adenocarcinoma—Gallbladder	1
Adenocarcinoma—Rectum	10
Pseudomucinous cyst—Ovary	2
Carcinoma—Prostate	1
Generalized abdominal carcinomatosis	4
Polyp	1
B. *Small Bowel*	
Adenocarcinoma of the ileum	4
Generalized abdominal carcinomatosis	4
Fibroid tumor of ileum	3
Pelvic carcinoma	2
Adenocarcinoma of duodenum	1
Fibroid uterus	1

neoplasms, only 1 was due to a benign tumor (a rectal polyp). However, of 15 tumors causing small bowel obstruction, 4 were benign—3 fibroid tumors of the ileum and an extraluminal obstruction caused by a uterus containing large fibroids.

As noted, the mortality rate ordinarily is higher in large bowel obstruction, due to the larger number of malignant processes which act as the etiologic factor and the frequent chronicity of the process before obstruction becomes complete. Of the 159 cases of large bowel obstruction recorded, 62 (39 per cent) were due to neoplasms and 61 of these were due to malignant neoplasms. On the other hand, in 1128 cases of small bowel obstruction only 15 (1.3 per cent) occurred as a result of neoplasms and only 11 (1.0 per cent) as a result of malignant neoplasm. Of the total cases of obstruction due to neoplasm, 80.5 per cent were in the large bowel and the remainder were in the small bowel.

Congenital anomalies. Excluding hernias, which have been considered, congenital anomalies accounted for 5.3 per cent of the total (69 cases). Of these, 31 (45 per cent) were due to congenital malrotation, 5 due to adhesive or peritoneal bands, and 2 due to annular pancreas. The incidence and management of obstruction in infants and children were presented in a previous paper.[3]

Volvulus. This group accounted for 4.4 per cent of the total cases, 3.5 per cent being in the large bowel (sigmoid) and 0.9 per cent in the small bowel. This represents a higher incidence than usually is reported elsewhere in the United States, and possibly is due to the heavy preponderance (80 per cent) of Negro patients included in this series. The surprisingly frequent occurrence of this condition in this segment of the population of Memphis was noted by Pool and Dunavant.[1]

Miscellaneous. Diverticulitis was the cause of obstruction in 3.9 per cent of the cases. Intussusception comprised 3.2 per cent of the total, or 42 cases, 40 of which were of the ileocolic type and 2 of which were in the large bowel. Of these 42 cases, 30 were in children under 1 year of age. Four patients with mesenteric thrombosis were admitted, all of whom died. There were 2 cases of gallstone obstruction with no deaths. (Incidentally, one gallstone measured 4 x 10 cm. in size.)

RELATION OF DURATION OF SYMPTOMS TO MORTALITY RATE

The duration of symptoms was ascertainable in 1188 of the admissions and these data are presented in table IV. In cases of small bowel obstruction there was a definite correlation between the length of symptoms and the mortality rate. Patients with symptoms of 24 hours or less prior to treatment exhibited a mortality rate of 9.5 per cent; those with symptoms longer than 96 hours had a mortality rate of 23.6 per cent.

The gross mortality rate in large bowel obstruction was greater (27.5 per cent) than that in small bowel obstruction (14.9 per cent). This was due in part to the fact that the former occurred more commonly in the elderly patient who was less able to withstand the physiologic alterations produced by the obstruction and who commonly had associated pathologic conditions such as heart or renal failure. In addition, malignant tumors with accompanying debility constituted a major cause of colon obstruction. Although it might appear, upon inspection of table IV, that the duration of obstruction had no direct correlation, or even had an inverse correlation, with the duration of symptoms, such a conclusion

TABLE IV

Length of symptoms before admission

Duration	Lived	Died	Per Cent
Large Bowel			
24 hours and less	23	7	23.3
48 hours and less	7	5	41.6
72 hours and less	8	1	11.1
96 hours and less	8	2	20.0
96 hours and more	49	21	30.0
Total	95	36	27.5
Small Bowel			
24 hours and less	435	46	9.5
48 hours and less	127	18	12.4
72 hours and less	90	20	18.1
96 hours and less	69	19	21.6
96 hours and more	178	55	23.6
Total	899	158	14.94

would be erroneous. Partial obstruction often had existed for a considerable period of time. Complete colon obstruction, which represents closed loop obstruction if the ileocecal valve is competent, is an extremely serious and poorly tolerated condition.

IMPORTANCE OF EARLY OPERATION

Of the 1240 patients whose hospital record showed accurately the length of hospitalization before operation, 612 (49.3 per cent) were operated upon within 12 hours after admission, with a mortality rate of 11.9 per cent. The remaining 628 patients were not operated upon in less than 12 hours; they were either operated upon later or were treated conservatively, with an over-all mortality rate of 19.4 per cent. Of these 628 patients, 350 were treated conservatively; that is, with gastrointestinal suction, parenteral fluids, and other supportive therapy as indicated (mortality rate 20.9 per cent). The remaining patients, who were operated upon after 12 hours' hospitalization, had a mortality rate of 17.6 per cent. It should be clearly stated, however, that conservative therapy was at times employed in the hope that preparation would enable the patient to survive operation, which because of the patient's condition never could be done.

These figures re-emphasize a point of critical importance. *The promptness with which the diagnosis and therapy are effected is of the utmost importance in reducing the mortality rate in cases of intestinal obstruction.* In small bowel obstruction, a delay of three days in hospitalization and treatment increased the mortality rate 250 per cent. A three day delay in operation after hospitalization was associated with an increase in mortality rate of 50 per cent, although in some patients operation offered little hope at any time. The conservative plan of therapy should be selected with caution and continued vigilance, for the mortality rate was 20.9 per cent in these patients. It again will be recalled, however, that included in this category were those patients admitted to the hospital in such severe condition that operation could not be undertaken immediately. Many of these patients died soon after admission, sharply increasing the mortality rate

TABLE V

Effect of bowel resection upon mortality rate

	Lived	Died	% Mortality
Small Bowel			
Not resected*	880	133	13.13
Resected	78	27	25.72
Large Bowel			
Not resected	104	34	24.6
Resected	15	6	28.5

* 350 of these were treated conservatively, 73 of whom died.

for this category. Notwithstanding, early operation results in a diminished mortality rate from intestinal obstruction.

EFFECT OF RESECTION OF BOWEL UPON MORTALITY RATE

In this series of 1287 patients it was necessary to resect portions of the bowel in 126 patients. Patients with small bowel obstructions who had resections had a mortality rate of 25.72 per cent (table V). This is to be contrasted with the uncorrected over-all mortality rate of 15.5 per cent.

What was the incidence of late complications following side-to-side anastomosis? Bowel resection was done with survival of the patient in 67 instances. The following types of anastomoses were made: 38 side to side anastomoses of the small bowel, 14 end to end anastomoses of the small bowel, and 15 end to end anastomoses of the large bowel. No early complications occurred in this series as the result of a failure of either type of anastomosis so far as could be determined from the records. There were no late complications from dilatation or *blowout* of the closed ends of bowel in the side to side type of anastomosis. Although the current trend is toward the end to end type of anastomosis, in our experience the side to side type of anastomosis has been satisfactory.

SUMMARY

A review of the etiologic factors and mortality rate in 1287 admissions to the hospital for intestinal obstruction is presented. The uncorrected over-all mortality rate was 15.5 per cent.

Adhesions (42.9 per cent), hernias (31.0 per cent), and tumors (5.9 per cent) accounted for 80 per cent of the total cases. Right indirect inguinal hernia was the cause of obstruction more than twice as often as left indirect inguinal hernia.

No particular complications following side to side anastomoses were recorded.

The crucial importance of early operation in diminishing the mortality rate due to intestinal obstruction once again is emphasized.

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OBSERVATIONS IN HAWAII ON SURGERY OF THE BILIARY TRACT*

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These recorded observations which are available for study are based upon 948 operations involving the biliary tract done by the staff of the Straub Clinic.

In Hawaii the opportunity has presented itself, over the years, to observe racial differences as related to disease and in previous publications attention has been drawn to these observations.[6, 7, 8, 9] For instance, it has been noted that cancer of the stomach is far more common in the Japanese race than in other races, particularly in the male. *Cancer of the breast in Japanese women in the past has been rare and this has been attributed most commonly to the fact that Japanese women usually nurse their children for much longer periods of time than other races. In more recent years, the incidence of this disease seems to be on the increase and this may be due to the Japanese adopting western habits regarding infant nursing.* Cancer of the skin is rare in all Oriental races. Diseases of the thyroid were rarely seen in any form some three or four decades ago but over the ensuing years, this picture has completely changed so that now all types of thyroid diseases, with the exception of toxic nodular goiter, are of frequent occurrence. Prostatic hypertrophy and prostatic cancer occasionally are seen in Orientals but relatively rarely. While sufficient studies have not been made to determine the reasons for these racial differences in disease, it would seem that it largely is a matter of racial predisposition and habits rather than local environmental conditions in Hawaii.

The question of the frequency of gallstones occurring in the various races in Hawaii was investigated, probably extensively enough to arrive at some fairly accurate conclusions. During the years 1951, 1952 and 1953, a diagnosis of gallstones was made 768 times, combining the statistics of The Queen's and St. Francis Hospitals. During this time, there were 72,863 admissions, an over-all incidence of patients with gallstones to patient admissions of 1.05 per cent. Japanese and Filipinos had the lowest incidence of gallstones to admissions, being approximately .6 per cent, Koreans 1.15 per cent, other races averaging about 1.6 per cent. Any opinions arrived at as to the cause of this variation of incidence in these races is entirely conjectural in nature, but I am inclined to believe that diet and physical build play an important role.

The subject of biliary tract disease is too large to permit discussing more than a few of its aspects which to me seem worthy of emphasis at this time. The jaundiced patient frequently presents problems in diagnosis that are difficult to clarify, and this subject, a lengthy one in itself, will be discussed at a later time.

It has been my observation that many of us fail to recognize or we overlook the symptoms that may be produced by gallbladder disease. I have been chagrined over such an oversight on more than one occasion and I hope these errors

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have made me more acutely aware of such possibilities. The patient with an acute gallstone colic or the individual with jaundice rarely fails to receive adequate investigation, but all too frequently individuals with symptoms of coronary heart disease, those complaining of mild indigestion and those with a typical history compatible with a gastric or duodenal lesion fail to receive any attempt at gallbladder visualization. No individual with an unsolved complaint of abdominal distress has had an adequate investigation unless an effort has been made to visualize the gallbladder. In my opinion, no individual suspected of having a gastric or duodenal lesion, unless there is hemorrhage or perforation, should have a gastrointestinal study until the gallbladder has been investigated. This should be done first because the ingestion of barium may, and frequently does, interfere with the gallbladder shadow. The gallbladder, the duodenum and the pyloric end of the stomach are too closely associated anatomically to permit accurate symptomatic differentiation when involved by a lesion. It has been my unhappy experience, as I am sure it has been that of others, to encounter an undiagnosed duodenal or gastric lesion when doing a cholecystectomy for stones. What surgeon among us has not in the course of a laparotomy palpated a gallbladder containing stones which were previously unsuspected. It should be remembered that small stones or papillomas easily diagnosed by visualizing the gallbladder preoperatively, are easily overlooked or impossible to detect by palpation of the gallbladder. Also lesions in the stomach or duodenum recognizable by roentgenol study may be overlooked by examination at the time of laparotomy for other indications.

Figure 1 represents a patient, recently operated upon, in whom a gallbladder visualization revealed a non-functioning gallbladder in association with a large ulcerating lesion of the stomach. The patient's symptoms at the time of examination suggested a gastric lesion and, true to Oriental custom, he did not reveal that some 10 years previously, he had had symptoms of gallstone colic, and at that time an attempt to visualize the gallbladder had been unsuccessful. True, no difficulty in recognizing this diseased gallbladder at the time of operation would have been encountered, but it always is better to know what the surgical problem to be dealt with is *before*, rather than *after*, making the incision.

The question always arises, when the abdomen is opened for some other condition and on palpation the patient is found to have gallbladder calculi; or from previous examination the patient is known to have calculi; whether or not one is justified in removing the gallbladder. This decision does not lend itself to solution except as encountered at the operating table. However, on deciding this point, it is well to remember that complications from such a gallbladder may occur in the postoperative course that would have been better to prevent by doing a cholecystectomy at the time of the original laparotomy.

In 1942, I was called in consultation to see a Japanese man, age 41 years, who seven days previously had been operated upon through a McBurney incision and an acutely inflamed appendix was removed. The clinical diagnosis had been verified by microscopic study showing an appendix involved in an acute inflammatory process with areas of ulceration and gangrene. On the fifth postoperative

FIG. 1. Association of gallstones and gastric ulcer

day, he began for the first time to complain of right upper quadrant pain and distress associated with fever and leukocytosis. Examination revealed localized tenderness and rigidity over the gallbladder area and at operation an acutely inflamed gallbladder was found. The surgeon elected to drain the gallbladder. No stones were found. The patient made a good recovery and has remained free of symptoms since.

A woman, age 82 years, was found to have gallstones at the time of operation for acute intestinal obstruction due to adhesions, probably secondary to a previous inflammatory reaction about the gallbladder which was found to contain gallstones. The obstruction was relieved and the incision closed without removing the gallbladder because the patient's age and known cardiovascular disease and hypertension placed her in the category of a poor surgical risk. Three weeks later she developed the characteristic picture of acute cholecystitis. At operation an acutely inflamed gallbladder was found that had perforated and had been walled-off by omentum. The gallbladder was removed and numerous stones also were removed from a much dilated common duct. Surprisingly the patient made an uneventful postoperative recovery and died about two years later from her cardiovascular disease.

An elderly Chinese man, age 76 years, was passing through Honolulu on his way to the Orient when he developed pain and tenderness in the right upper quadrant associated with fever and leukocytosis. At operation bile peritonitis was found and drained. Six weeks previously in the mainland of the United

States, he had had a gastric resection for a benign ulcer of the stomach. He died one week following the drainage operation from myocardial infarction. An interesting finding at the autopsy was a stone in the cystic duct of the gallbladder, and there was evidence of a healing perforation in the fundus. No doubt the gallstone by obstructing the cystic duct had initiated the train of events leading to this man's death.

Schwegman[5] and associates at the University of Pennsylvania Hospital have called attention to the occurrence of acute cholecystitis following operation for unrelated disease in 17 patients in the 23 year period previous to 1953. Glenn[4] at New York Hospital, over a 14 year period, had 17 such complications under similar conditions. In 3 of Schwegman's patients, cholecystitis was not recognized until autopsy; in 4 the gallbladder had perforated; in 4 there was gangrene without perforation; and in 9 the gallbladder was acutely infected. In 35 per cent of the patients, no stones were found in the gallbladder. In attempts to explain the tendency of disease of the gallbladder to flare up in the postoperative period, it has been postulated that operative trauma associated with the administration of narcotics results in stagnation and concentration of bile in the gallbladder. Since most frequently the symptoms of cholecystitis develop a day or two following the onset of oral ingestion of food, it is suggested that this act results in forceful contraction of the gallbladder resulting in plugging of the cystic duct by the concentrated bile or by stones. Needless to say, unless one is aware that the patient has a diseased gallbladder, symptoms of beginning cholecystitis in all probability will be ascribed to other reasons which much more frequently give rise to such symptoms. It is worth emphasizing that it is well to examine thoroughly individuals before subjecting them to surgery, and also to remember that regardless of the circumstances, one cannot *lightheartedly* close an abdomen that is known to contain a diseased gallbladder.

Many years ago, Babcock[1] called attention to the frequency with which myocardial diseases, and particularly anginoid pains, are associated with gallbladder disease, and it has been shown by clinical observation and autopsy studies since then that coronary artery disease and gallbladder disease frequently are associated. Walsh and associates[11] in 1798 autopsy studies found that the association of these two diseases was twice as common as was their occurrence found separately. Whether gallbladder disease predisposes to or aggravates coronary artery disease, or whether their frequent association is coincidental is, so far as I can determine, a point still in dispute by cardiologists. Personally I know of instances of individuals who led a life of semi-invalidism, because of having been diagnosed as having coronary artery disease, whose symptoms and invalidism have cleared up following the removal of a diseased gallbladder. It has been my further observation that pain from such calculi may be located on the left side along the costal margin, the cardiac area or left subscapular region. Pain in these areas has cleared up following cholecystectomy for a diseased gallbladder. Regardless of the underlying relationship of these two diseases, it behooves all of us who are interested in these problems to adequately examine the patient so as not to overlook either a diseased gallbladder or an ailing heart.

The question frequently arises as to whether or not it is always advisable to

recommend removal of the gallbladder which is found to contain stones but is giving little or no symptoms. I doubt if many gallstones are completely silent. Occasionally they are present and the host has little to remind him of their presence. It has been our custom to advise everyone who is an adequate physical operative risk to have such a gallbladder removed because, in common with other observers, we have seen serious complications arise when this advice was not heeded.

A 47 year old man was advised to have a cholecystectomy because of evidence of gallstones which was confirmed by roentgenologic studies. Approximately two years later, 48 hours after onset of severe abdominal pain with nausea and vomiting, the abdomen was explored. There was extensive fat necrosis in the region of the gallbladder and in the omentum. An acutely inflamed gallbladder containing stones was removed. After a very stormy postoperative course, which included subcostal drainage on the left of a large abscess in the lesser omental cavity, undoubtedly secondary to the acute pancreatitis, the patient ultimately recovered. Many such complications which are associated with recognized but delayed treatment of gallstones—some of them with a less happy outcome—could be cited. Acute cholecystitis, hydrops of the gallbladder, obstructive jaundice, hepatitis, cholangitis, pancreatitis, fibrous stricture of the common duct, intestinal obstruction and undoubtedly cancer of the gallbladder may result from delay in treatment of patients with gallstones. How much more satisfactory it is to recognize and remove the diseased gallbladder early than it is to attempt to cope with such complications as just mentioned. In evaluating an individual as to his or her operative risk, it also is well to remember that cholecystectomy in the hands of a competent surgeon with adequate facilities to carry out such a procedure is not a matter of serious portent. Therefore, the contraindications to cholecystectomy should not be drawn too finely. The mortality rate without complications should be less than 1 per cent and in some large series, it has been reported to be considerably less. It is in the presence of complications of gallstones, especially in elderly people, that mortality and morbidity rates following surgery assume considerable proportions. It also is worth remembering that gallstones usually occur in the age group where the passing years in general makes for a poorer surgical risk.

It has been my experience that the individual with a good gallbladder history whose roentgenologic studies show gallstones, or whose gallbladder fails to visualize after at least two attempts, will in the vast majority of instances have a satisfactory result following cholecystectomy. We have found telepaque, 3-(3-amino-2,4,6 triiodophenyl)-2 ethylpro-panoic acid, to be the most satisfactory material to use as a means of visualizing the gallbladder. One should be very skeptical about the value of cholecystectomy if the only evidence suggesting trouble is a poorly-filling or poorly-emptying gallbladder even though the history suggests gallbladder disease. Some observers seem to doubt that clinical improvement will follow removal of gallbladders that show only cholesterosis, known as the *strawberry* gallbladder. Much of the literature dealing with this subject has been accumulated from autopsy studies[3] and differs, I believe, from the surgeons'

approach to this problem. We do not advise removing gallbladders unless the patient has a history compatible with gallbladder disease and unless the gallbladder fails to visualize. Under such circumstances, we have found that the gallbladder usually shows, in addition to cholesterosis, small stones and frequently associated polyps. One is never sure preoperatively that a small filling defect in the gallbladder, as suggested by its constancy of location in the periphery, is due to a polyp or to a stone. Removing gallbladders that have one or even several small polyps as the only evidence of disease is justifiable because such lesions may be a precursor of malignancy, and I am quite sure that portions of such polyps at times may break off and cause intermittent obstruction of the cystic duct. Individuals from whom we have removed such gallbladders have been happy with the results.

For many years the question has been debated as to the advisability of using a T tube or some other form of drainage in the common duct that has been opened and in which nothing abnormal has been found. It has long been my practice to close such ducts snugly with fine atraumatic plain catgut bringing together the surrounding peritoneum over this area. Adequate drainage down to the stump of the cystic duct using, Penrose drains and a soft rubber tube or catheter, always is provided. This method of procedure has been used on many occasions, and I can recall no regrettable experiences. A smoother and a shorter convalescent period, I believe, has resulted from this practice. The only reason for placing a T tube in the common duct that, for all intents and purposes is free of disease, after adequate exploration fails to reveal anything abnormal in the common duct or with the opening into the duodenum is, from my point of view, to enable one to make a postoperative cholangiogram. Drainage is instituted following all cholecystectomies for bile certainly is better tolerated by the patient if it is in the dressing instead of being free in the abdominal cavity. Regardless of how carefully the cystic duct is ligated, there frequently is a considerable drainage of bile, in most instances, no doubt, resulting from small bile ducts being opened in the gallbladder bed.

For several years we have been attempting to establish the role that operative cholangiography should play in biliary duct surgery, but as yet our conclusions have not become stabilized. Judging from current literature and from first hand observation in clinics in various parts of the world, no unanimity of opinion exists as to the value of this procedure. I became more acutely aware of the possible value of this method of diagnosis, in selected cases at least, some time ago because of the following tragic experience.

A woman, age 55 years, with known gallstones of several months' duration, was, with reluctance on her part, persuaded to undergo surgery. Careful preoperative investigation led to the belief that she was a good surgical risk. The gallbladder containing stones was easily removed. The common duct was explored and found to contain several stones. After careful irrigating, scooping and probing, the ducts seemed free of calculi, a conclusion one can never reach with any degree of certainty. Much to my surprise a cholangiogram 10 days postoperatively revealed a calculus, as shown in figure 2. The wound was reopened

FIG. 2. Postoperative cholangiogram showing residual stone not found at time of exploration of common duct.

on the fifteenth postoperative day and the calculus was removed without difficulty. The patient left the operating room in good condition but several hours later, she went into collapse and died, the cause of which was not determined even at postmortem examination. It was my remorseful conclusion that had I done a cholangiogram on the operating table, this calculus would in all probability have been recognized and removed and the subsequent fatality would have been avoided. Since this time more frequent operative cholangiograms have been made, but the results have not by any means been universally satisfactory. The procedure is time consuming; for success it depends upon an exact technic; stones are not universally recognized, and it frequently is difficult to differentiate stones from air bubbles.

Comparatively recently the discovery of contrast media with greater radiopacity, which are excreted preponderantly in bile, has made it possible to visualize the biliary tract preoperatively in a large percentage of patients. Cholografin (3-amino-2,4,6-triiodo) benzoic acid with iodine content of 64.23 per cent has been found to have this property. It has the disadvantage of requiring intravenous administration and necessitates skin testing for sensitivity for safety. The dye is excreted promptly and rapidly, a relatively high concentration being obtained in the ducts in 20 to 30 minutes. If the gallbladder is present without cystic duct obstruction, it also is clearly visualized. The dye also is an indicator of liver function since in normal livers approximately 90 per cent is excreted by

this organ and 10 per cent by the kidneys. With liver damage, depending upon the extent, this ratio of excretion may be reversed so that no visualization of the biliary ducts is seen but good pyelograms are obtained. The ability to adequately visualize the biliary tree preoperatively no doubt will be of great aid in helping to rule out the necessity of exploring the ducts at the time of operation if no abnormality by this method of visualization is noted. The ability to quickly visualize the biliary system may be of help in arriving at a differential diagnosis when this seems desirable and when time seems to be an important factor. Such a method of examination also should be very helpful in ruling out the presence of common duct stones in the presence of the postcholecystectomy syndrome. We have found it necessary to begin taking X-ray pictures soon after injecting the dye and to continue at frequent intervals in order to obtain the most desirable degree of filling of the gallbladder and ducts. With too little concentration, the dye fails to visualize the lumen of the ducts and with too much concentration, it may obscure stones, particularly if they are small and also depending upon the degree of their radiopacity. Visualization of the biliary tract is not dependent upon the presence of the gallbladder, and gallbladders with poor powers of concentration, not visualized by the usual preparations, may be shown well by the use of cholografin. Figure 3 a shows an attempt at visualizing

FIG. 3 a

FIG. 3 a. Gallbladder not visualized by double dose of telepaque. b. Same patient. Gallbladder visualized by use of intravenous cholografin. c. Postoperative cholangiogram showing biliary ducts free of stones.

FIG. 3 b

FIG. 3 c

a gallbladder using a double dose of telepaque. Poor filling of the gallbladder is seen. Figure 3 b shows the same patient following intravenous injection of cholografin. The gallbladder is well visualized as are the hepatic and common ducts. Several filling defects in the common duct indicate the presence of stones. At operation a small shrunken gallbladder was removed. The common duct was considerably dilated but much to our surprise no stones could be found even after transduodenal exploration of the ampulla of Vater, and operative cholangiography indicated that the ducts were free. Subsequent visualization of the ducts by way of the T tube (Fig. 3 c) showed nothing suggesting stones. A few days before operation the patient had severe abdominal pain and the only explanation that seems logical is that the stones were passed at this time. Perhaps gas from the duodenum could have regurgitated up the common duct to give the picture suggesting gallstones, a possibility that I have not seen mentioned by other observers.

The postcholecystectomy syndrome—biliary dyskinesia—continues to be an entity which is poorly understood and difficult to diagnose with assurance. It occurs following cholecystectomy for stones, but more frequently following removal of gallbladders without stones in which study of the gallbladder by the pathologist fails to account for the patient's preoperative symptoms. If the patient has had stones removed previously from the biliary ducts, one's first thought is that a stone was overlooked or has since formed. The use of cholografin may be of great value in helping to decide this point. Figure 4 is a cholo-

FIG. 4. Cholografin study of extrahepatic ducts showing common duct stones following previous cholecystectomy.

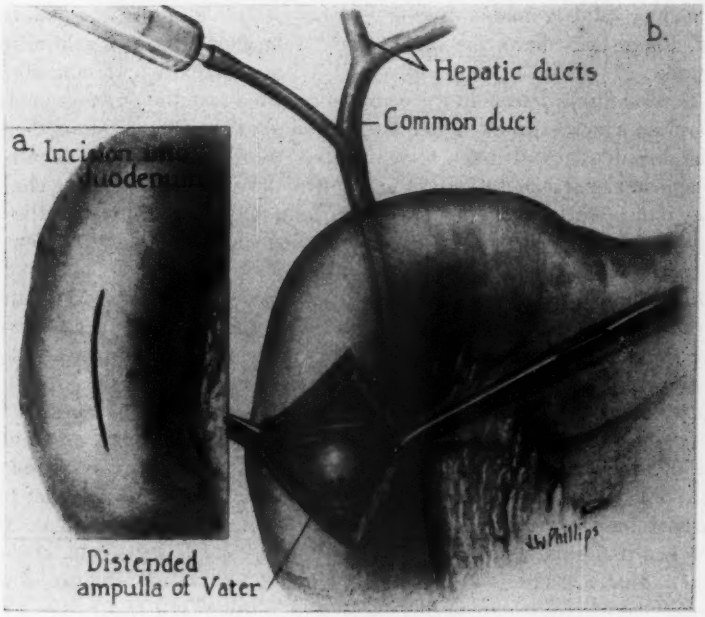

FIG. 5 a b

FIG. 5 a, b, c, d, e. Showing the procedure used in exploring the common duct in suspected cases of biliary dyskinesia and the type of sphincterotomy used when such a diagnosis is made. (Strode, J. E.: Biliary Dyskinesia from the Surgical Viewpoint, Ann. Surg. *117:* 198 (Feb.) 1943.)

grafin study of a patient who previously had gallbladder drainage for acute cholecystitis and later had a cholecystectomy for stones. Recently symptoms of abdominal distress and pain in the right upper quadrant suggested the possibility of recurrence of stones in the common duct or the postcholecystectomy syndrome. It seems evident that there are a number of stones in the common duct.

In a previous publication[10] the method we have used in dealing with patients suffering from biliary dyskinesia is described (figs. 5 a to e), and we have continued to use this method with gratifying results. Our cases have been few, numbering in all not over 12. These patients have for the most part been relieved of symptoms following sphincterotomy at the ampulla of Vater. From this experience, it would seem logical to conclude that the postcholecystectomy syndrome results from spasm of the sphincter causing increased intraductal pressure.

Our experiences with attempts to cause disintegration of common duct stones by the method advocated by Best and associates,[2] while limited, has not met with success. Patients are greatly distressed when this method is used and our experience has not encouraged us to insist that the attempt be long continued.

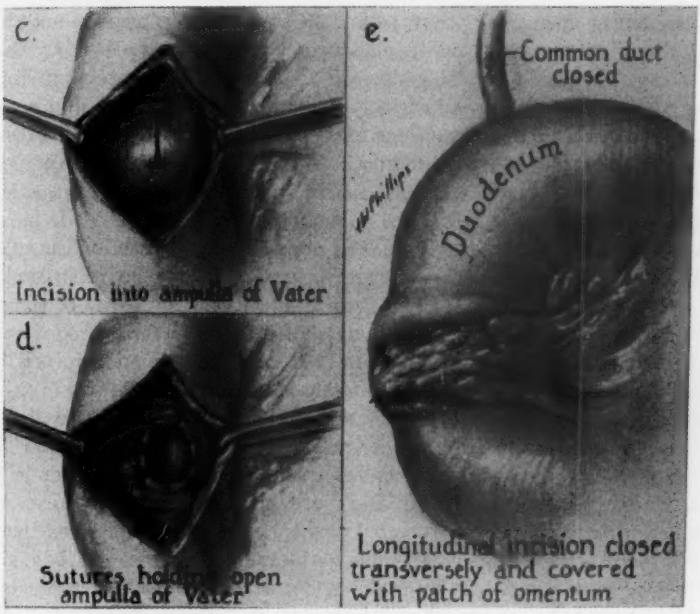

FIG. 5 c d e

It is hoped that further experimentation will yet bring forth a conservative method of removing such stones that will be satisfactory to all concerned.

ACUTE CHOLECYSTITIS

It has been my observation that many cases of acute cholecystitis will subside if given the opportunity. In which patients this favorable turn of events will occur, there is no method of determining without the test of time. In contradistinction to most cases of appendicitis, the tendency is for such inflammations to subside and relatively few such acute episodes involving the gallbladder go on to empyema or gangrene with perforation. Most gallbladders that perforate under such circumstances remain walled-off and few give rise to peritoneal infection. With the use of antibiotics, this possibility has become even more remote. The debate over the opportune time for surgical intervention in such patients continues and no doubt never will be settled to anyone's complete satisfaction. If the diagnosis seems certain, particularly if the patient is known to have gallstones and is an acceptable risk, it would seem advisable to remove the gallbladder immediately before inflammatory reaction has become so pronounced as to make identification of the structures around the cystic and common ducts a difficult matter. The tendency generally is to procrastinate hoping that the acute symptoms will subside and surgery can be done at a more opportune time when structures are more easily identified and, particularly when indicated, that the common duct can be explored with greater safety. Many

observers advise operating within the first 48 hours if symptoms are not abating, and if there is increase in spontaneous pain and tenderness—leukocytosis and fever being the most dependable signs that the attack is progressing. It is surprising at times to find how extensively the gallbladder is involved in a gangrenous process even though symptoms have been of short duration. It also is an interesting observation that frequently no stones are found even in a gangrenous gallbladder and occasionally in association with empyema. It always is most desirable to operate early before the inflammatory reaction has made identification of structures difficult or at some later period when the reaction has subsided, but it frequently becomes necessary to intervene at a time when such ideal conditions do not exist. Under these circumstances, one should not be committed to the belief that the gallbladder should be removed in the orthodox manner. It may be advisable to begin dissection in the fundal area and proceed toward the cystic duct in order to more adequately identify the structures in this area. If the patient is a poor operative risk and structures are difficult to identify, it may be wise to drain the gallbladder and do a more definitive operation at a later date. It may be good judgment in certain cases, if the patient is elderly and a poor surgical risk, to do nothing more unless compelled to do so. I know a number of patients who were treated in this manner who completed their span of life without further troubles referable to the gallbladder. Briefly, then, it would seem logical to advise people with gallbladder calculi to have a cholecystectomy for fear, among other things, of the development of acute cholecystitis. If signs of acute cholecystitis develop and the patient is seen in the first few hours, immediate operation should be advised. After this period, if the attack seems to be subsiding, the patient should be observed with the hope that an elective operation can be done later under more favorable conditions. If the attack seems to be progressing or not improving, surgical intervention is indicated. Many and varied mitigating circumstances frequently arise to influence one's decision under certain circumstances, so each case must be judged on its merits.

No one should ever write on the subject of biliary surgery without emphasizing the potential dangers associated with cholecystectomy. Injury of the extrahepatic ducts is an extremely serious complication from which the patient all too frequently never recovers even in the hands of those most expert in repairing such injuries. Adequate exposure of the operative field, meticulous identification of the structures about the cystic duct where so many anomalies occur, and proper training of surgeons before attempting this type of surgery, are the measures to be used in keeping such unfortunate complications at a minimum.

CONCLUSIONS

In conclusion I would like to emphasize the fact that gallstones are of common occurrence particularly in women from the fourth decade of age and beyond. Both internists and surgeons should be acutely aware that, while their presence usually is manifested by symptoms characteristic of this disease, frequently such is not the case. For this reason it is advisable to resort to more frequent use of

gallbladder visualization in individuals who have abdominal complaints, particularly so if symptoms are not otherwise easily explained by other findings. It is advisable in all individuals in the gallstone age, who are undergoing an elective abdominal operation, to have a preoperative gallbladder investigation for the reasons previously mentioned. It has been emphasized that while gallstones may be silent, they never are to be discredited and, all things being equal, they are better in a specimen bottle than in the patient's abdomen. And, let me again urge that all surgeons who operate upon the biliary tree should be adequately trained before they approach these problems. The potential dangers to the unsuspecting patient are too great to condone the all too frequent attempts of the tyro to cope with these problems.

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BACK DISABILITY

1930 to 1955

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The past quarter century has disclosed dramatic progress in the understanding and treatment of many medical problems, and of few is this more striking than in disabling back pain. To have shared in the successful attack on this perplexing problem has been a satisfying experience—many times disappointing in our discouraging errors—but also many times rewarding in our successes.

Let us review for a moment the dilemma of the patient with a disabled back 25 years ago. Because of poor understanding of the underlying pathology, many diverse explanations were given regarding the cause of back and sciatic pain such as foci of infection, sacroiliac strain, retroversion of the uterus, ptosed kidneys, prostatic disease, and even allergy. Accordingly, many different treatments were offered with, of course, inconsistent results. Patients commonly went from doctor to doctor, seldom receiving any lasting relief. Particularly unfortunate was the plight of the patient whose livelihood was threatened by a chronic back disability. Since cures were rare, he not only had to endure his pain, but also to contemplate a bleak future as a wage earner. In addition, insurance and compensation coverage at that time was woefully inadequate. As in other phases of medicine, the psychosomatic aspects of the problem were too often neglected and many patients had to cope with emotional difficulties in addition to physical and economic stress. It is little wonder that the back problem so often perplexed not only the victim, but all others concerned.

The first real light was shed on this problem in 1933 by Mixter and Barr[4], who presented an epochal report of cases of rupture of an intervertebral disc causing low back and sciatic pain. This marked the beginning of the recognition that a damaged intervertebral disc is the usual cause of disabling back or sciatic pain and started us on a new path in the handling of this problem. It was theorized that, if the ruptured disc was the pain producing lesion, then its surgical removal would control the pain. As a result, operations for the removal of ruptured discs became widespread. While the severe pain usually is relieved by this procedure, a significant number of patients are unable to return to their former occupations because of physical limitations. In an effort to improve matters, dorsal fusion sometimes has been added to the surgical procedure, usually carried out by a neurosurgeon-orthopedist team but the results hardly seem to justify the increased surgery and the divided responsibility.

As time has passed, we have been able to point to a remarkable increase in our knowledge and understanding of the pathology of intervertebral disc lesions and the mechanism of pain production. Unhappily, however, our ability to

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handle these problems has not always kept abreast of this understanding, and it has been only in relatively recent times that improvements in technic have made practical procedures which actually repair the basic lesion.

In reviewing the problem, let us consider the fundamental cause of intervertebral disc lesions and the manner in which pain is produced. When the integrity or well-being of tissue is threatened, pain, which is nature's defense signal, results. Physical stress appears to be the most frequent threat to the well-being of the tissues of the spine. The normal functions of weight bearing and motion subject the spine to a certain degree of physical stress, and additional strain often results from injury or the performance of arduous tasks. Unlike the vertebrae, which can withstand all but violent stress, the intervertebral discs, which support the vertebrae, frequently succumb to physical strain. It is the nucleus pulposus within the disc which normally supports the weight above. When excessive strain causes this to rupture, the load then must be borne by the residual portion of the injured disc, leading to progressive disintegration and instability. It is the consequent strain on tissues, beyond their comfortable tolerance, which results in back pain. With disintegration, the disc becomes thin, protrudes about the margin, and portions of it may become detached and extruded. If the nerve root, which occupies close quarters in its position between the disc and the ligamentum flavum, becomes compressed, pain will radiate into the root distribution. Hence, we note that back pain is caused by local stress on tissues from loss of support, while radiating pain is due to nerve root compression and irritation. Further, protective spasm may add aching muscles as a secondary mechanism of discomfort.

In considering a patient with these symptoms, it is well to keep in mind some of the far reaching complications of his problem. If he is handicapped by his physical disability in the pursuit of his normally profitable activities, he becomes concerned not only by the local distress, but also by the threat which is presented to his economic security. As his status becomes progressively more insecure, defensive responses become involved and various emotional disturbances may develop. Hence, we see some patients who experience the gamut of distressing pain, physical restriction, and emotional reaction with a resulting disability so complicated that rehabilitation is difficult, if not impossible. Clearly, then, if we are to rehabilitate these patients, an understanding of the total problem is required. It must be pointed out, however, that a patient can never become rehabilitated until his pain is relieved sufficiently to allow him to return to useful work. In this connection, we cannot place too much stress on the importance of the time element, for the length of the period of disability is basic to the patient's problem. Patients who are allowed to remain disabled for too long a period of time may become victims of chronic stress and remain emotional invalids. Thus, our efforts to shorten the period of disability will be very rewarding.

Any rational treatment of ruptured intervertebral discs must be aimed at the reduction of physical stress upon the injured tissues about the spine with time allowed for healing. The fact is well established that rest, along with conserva-

tive symptomatic measures such as traction, bed boards, and back supports will be sufficient to handle most minor back pain, as well as some of the major problems. Additional benefit may be offered by the judicious use of heat, massage, and other forms of physical therapy.

Most of the patients who recover on such a conservative regime do so within the first few days or weeks. If, however, several weeks elapse and the patient has shown no significant improvement the advisability of surgery should be considered. Without surgery, most of these patients may expect to remain chronically disabled, although a few will improve somewhat. All of them are likely to have increased pain at any time that the spine is subjected to physical stress. The general pattern is one of improvement with rest and inactivity and of aggravation with increased activity. Bearing this pattern in mind, and giving due weight to such factors as the patient's age, the degree of disability, and the physical demands which may be made of him, we may be in a position to advise the patient. The decision is one of whether the best interest of the patient will be served by his accepting the pain and disability and living within the necessary physical restrictions or by his undergoing surgery with the expectation that his pain and physical restrictions will be greatly reduced or eliminated.

DIAGNOSIS

It naturally follows that any sound planning for surgical correction must be based upon accurate diagnosis. It is not enough merely to decide that a disorder of the spine exists, or even that the disturbance is due to a disintegrated or protruding disc. It becomes necessary to determine which disc or discs may be involved and what may be required to control the pain. In addition to the usual physical and neurologic examinations, the surgeon relies upon roentgenography, myelography, and discography—as indicated—for aid in making a final diagnosis.

Roentgenographic visualization of the spine may suggest disc disorder by demonstrating changes such as hypertrophic reaction about the margins or narrowing of the disc. While these changes often have been referred to as some form of spinal arthritis or spondylitis, we believe that they merely represent the tissue responses to the chronic strain of function and injury and, therefore, are indicative of a strained or damaged disc. They occur more commonly in the lower cervical and lower lumbar regions where the physical stress of weight bearing is greatest. In addition, their incidence appears to bear a direct relationship to the degree of physical stress to which the spine has been subjected. Interestingly enough, we have seen spurs develop about the margins of a ruptured disc, and then watched them disappear when the strain has been relieved by surgical fusion, just as callus formation has been observed to disappear when healing about a fracture has been complete. The process, therefore, is reversible. We would add that it seems probable that any disease process which impairs the well-being of the supporting tissues would render them more susceptible to physical stress. These roentgenographic changes about the disc, then, probably indicate simply the reaction of the disc to *wear and tear*. In interpreting these

changes, it must be kept in mind that they require some time to develop and, therefore, their existence implies that the lesion is one of long standing. While such changes point suspiciously toward the corresponding disc as the origin of pain, they do not necessarily indicate that major disintegration of the disc has occurred.

While roentgenograms of the spine are helpful, the use of radiopaque dye usually is necessary for the more accurate diagnosis required in planning corrective surgery. Examinations using contrast media are of considerable value, but it must be remembered that their use carries a small, although definite risk. In considering such studies, therefore, the gains should be weighed against the risk involved. Further, the indiscriminate use of these procedures merely to establish a diagnosis of ruptured intervertebral disc or for medico-legal evidence is to be condemned.

Myelography, which demonstrates the configuration of the thecal sac, will localize a protrusion of the disc which causes a significant indentation into the subarachnoid canal, but the absence of an observable defect does not exclude the possibility of an internal disintegration of the disc or a small or laterally placed disc protrusion. It is estimated that the results of myelography will be negative or inconclusive in from 30 to 40 per cent of patients in whom a ruptured disc exists. Myelography, therefore, is of considerable value, but it, too has definite limitations as a diagnostic aid.

A newer, and perhaps more accurate diagnostic study is the discogram. This is obtained by injecting the central nuclear regions of the suspected disc with diodrast. In the intact discs, only a minimal quantity of dye can be injected and roentgenographic visualization shows it to be confined to the normal nuclear area. When the interior is disintegrated, a much larger quantity of dye can be injected and the roentgenograms may show the dye to be widespread throughout the disc structure. This test is proving to be of considerable value in demonstrating the cause of chronic, disabling back pain when all other findings and tests are negative or inconclusive. Discography is used widely in Sweden where it was introduced by Lindblom[3], but it has not been generally accepted in this country. Those opposed to its use fear damage to normal discs and this possibility must be considered. Cloward[2], who has used it extensively, states that he is unaware of the occurrence of any proved complications. Since it offers a means of establishing a definite diagnosis in the majority of the cases in which all other diagnostic aids are inconclusive, its value cannot be dismissed lightly. Judgment, therefore, should be exercised in its use and again the gains weighed against the risks (fig. 1).

TREATMENT

Once an intervertebral disc lesion has been accurately localized, there remains the problem of selecting the most appropriate surgical procedure. A number of technics are being employed currently for the relief of pain in these lesions. As we mentioned earlier, the surgical procedure which has been most generally accepted is the simple removal of the involved disc. This procedure decompresses

the crowded nerve roots to a large extent and can be expected to relieve most of the radiating sciatic pain in the majority of patients. It cannot be relied upon, however, to relieve back pain because the underlying instability is not corrected. Likewise, it does not relieve root irritation which may result from the instability. Following this procedure, many patients continue to be partially disabled because they still experience pain whenever certain physical limitations are exceeded. The results, therefore, have not been consistently satisfactory. This operation has its greatest field of usefulness in the control of unilateral sciatic pain in a patient who is in a position to restrict his activities without undue sacrifice. It has the advantage of being a much less radical procedure than one which includes stabilization about the disc and the convalescence is proportionately shorter.

For the patient who is disabled from back or sciatic pain and who is not in a position to limit his activities, we must look to a procedure which corrects the instability as well as decompresses the nerve roots. The logic of stabilization has long been recognized. Dorsal fusions of the laminal arches were done for

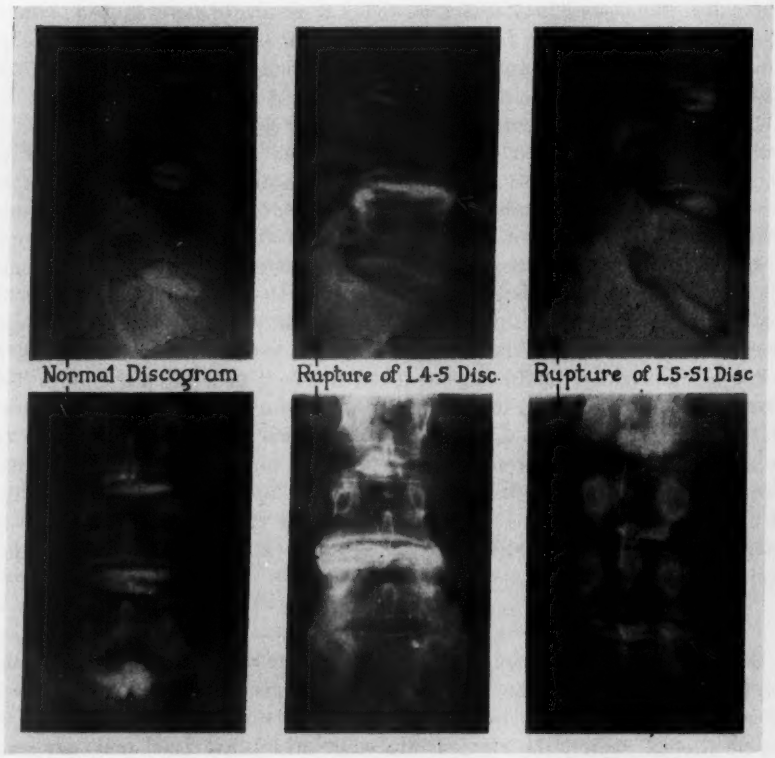

FIG. 1. Discograms obtained by the injection of diodrast into the nucleus pulposus

years before the ruptured intervertebral disc was recognized and, in fact, relieved pain in some patients. Too often they failed, however, because the roots were not decompressed and because fusion of the dorsal structures alone is not adequate to correct instability between the vertebral bodies. It has been realized for some time that to fuse the vertebral bodies about the injured disc would constitute the best means of supporting the unstable segment, since this is the site of the lesion and the center of the weight-bearing axis. Many years, however, have been required to advance from the recognition of the logic of interbody fusion to its practical accomplishment because of the technical difficulties involved in such a procedure. The first practical technic for interbody fusion has been developed by Cloward[1] over the past decade and involves the use of preserved refrigerated bone and specially designed instruments. One of us, (E. W.), has been employing various technics of interbody stabilization since 1943, using metallic and plastic devices as well as bone. In the course of time, we have come to agree with Cloward that only bone can be depended upon for lasting stability and have used his technic essentially since 1950. We have modified it to some extent and have added a prop-latch graft between the spinous processes to attain a more secure dorsal support (figs. 2, 3, 4).

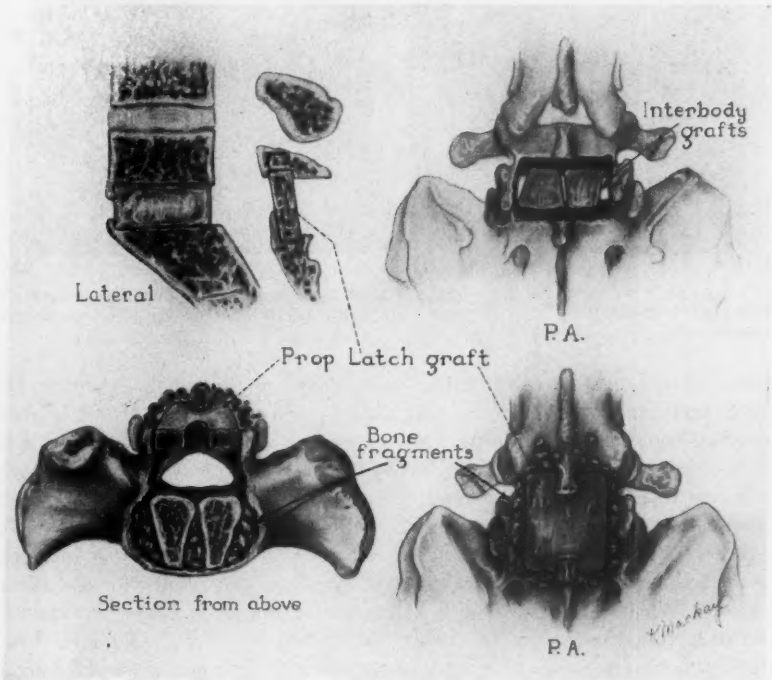

FIG. 2. Illustration to show the technic employed to obtain anterior interbody and dorsal interlaminar fusions.

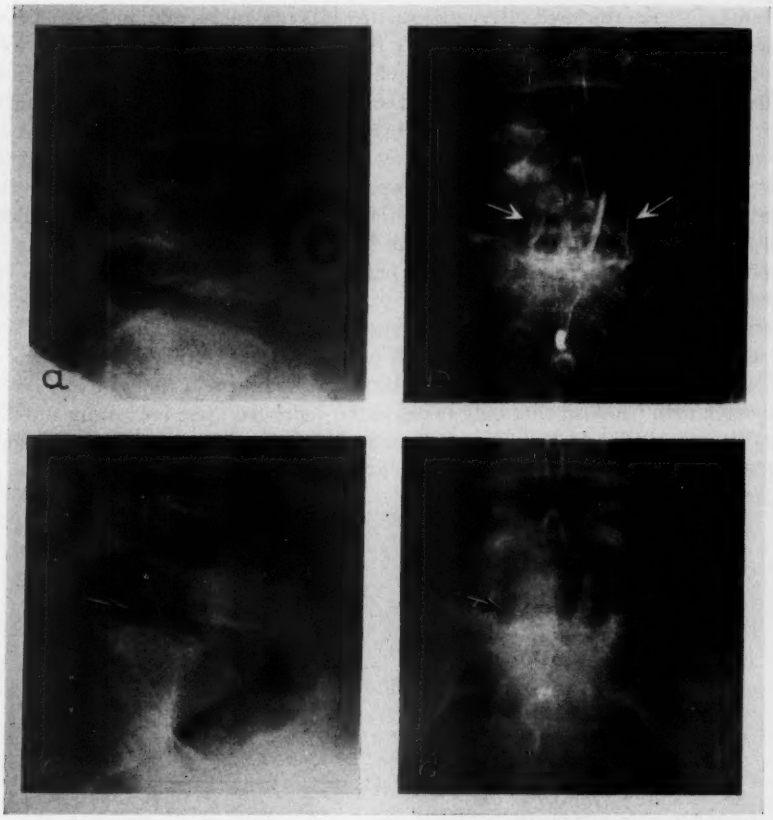

FIG. 3. (a) A preoperative film of case of rupture of lumbosacral disc. (b) Immediately following interbody insertion of iliac grafts. (c) and (d) Demonstrate progression of fusion three months following surgery.

The procedure, as we execute it, calls for the radical removal of the disc, after which grafting is done by removing the cortical end plates of the adjacent vertebral bodies and interposing grafts of bone taken from the liiac crest. While the vertebrae are forced apart, the bone plugs are seated with sufficient snugness to insure their retention, to sustain the width of the disc space, and to offer immediate structural stability. The facets and laminal arches then are grafted dorsally with bone fragments and finally a prop-latch *H* graft is interposed between the spinous processes, thus insuring adequate stability for the affected segment. When this procedure is satisfactorily done, no brace or support is necessary following surgery and the patient may be permitted out of bed as quickly as the postoperative discomfort permits. The ends of diminishing the period of disability and of hastening rehabilitation are greatly served by early

FIG. 4. (a) Preoperative film, rupture of lumbosacral disc. (b) Five months following interbody fusion. (c) Five years after surgery. Note replacement of disc space by bone and disappearance of proliferative spur.

ambulation and continued activity. It now is expected that the patient may return to limited work in from two to three months and we further expect that ultimately he will be able to perform full physical activities without restriction. The improved results of this procedure over the more conservative disc removals have encouraged us to resort to the stabilization procedure in an increasingly higher percentage of patients. Technically, the operation remains a difficult and time-consuming one, but we are compensated for our increased efforts by the rewarding end result.

SUMMARY

It has been deeply satisfying to see chronically disabled patients rehabilitated for heavy labor. It cannot be expected that every patient will be rehabilitated by any procedure, but the correction of instability by interbody fusion certainly is a significant step forward, and in our opinion constitutes the most important advance since the discovery of the ruptured disc. Improvements in technic which will simplify the procedure and improve the effectiveness are sure to come. To this end we will continue to dedicate our energies and our best efforts.

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POSTOPERATIVE PSEUDOMEMBRANOUS ENTEROCOLITIS

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The baffling complication of pseudomembranous enterocolitis which may follow any major surgical procedure will tax the diagnostic and therapeutic ingenuity of the attending surgeon. The diagnosis often is made at necropsy after failure of many therapeutic efforts. Death occurs in a very high percentage of those patients with this pathologic entity. Probably the first report of death from postoperative pseudomembranous enterocolitis was that of Finney[3] in 1893. He described a patient upon whom he had done a gastroenterostomy, but who died on the fifteenth postoperative day of severe *diphtheritic* colitis. Even though it more frequently follows surgery of the colon[8] it has been observed to follow operations on the gallbladder, uterus, brain, stomach, and breast.[1] The clinical and pathologic aspects of the disease have been described by Penner and Bernheim,[7] Dixon and Weisman,[1] and Kleckner, Bargen, and Baggenstoss.[6]

In 1939, Penner and Bernheim[7] reported acute ulcerative or diphtheritic colitis in 40 patients at autopsy from Mount Sinai Hospital in New York City. Shock was a common factor in these patients, and similar lesions were reproduced in animals in which shock had been produced by various means. These investigators stressed the importance of early diagnosis and intensive treatment of shock and diarrhea.

In 1948, Dixon and Weisman[1] reported the cases of 23 patients with acute pseudomembranous enteritis or enterocolitis complicating intestinal surgery who were observed during a 7½ year period beginning in 1940. They found no significant pathogens. The picture was that of rapid and progressive circulatory collapse with early death. Other symptoms were abdominal pain and distention, vomiting, fever, and diarrhea. In 21 of these 23 patients the disease was verified at autopsy, and the 2 survivors later were observed to have typical membranous casts in the feces.

In 1952, Hilsabeck and Dixon[4] reported the case of a patient who was suspected of having postoperative pseudomembranous enterocolitis following colon surgery who apparently responded to prompt and intensive antishock therapy. She later died of a pulmonary embolus, and the characteristic bowel lesions in the process of healing were demonstrated at autopsy.

Kleckner, Bargen, and Baggenstoss[6] in 1952 reviewed the records of the Mayo Clinic for the years 1940 to 1950 and found 14 cases of pseudomembranous enterocolitis which were not preceded by an operation. In 7 of the 14 patients the lesions were considered to be the immediate cause of death. In the other 7 patients it was not considered the cause of death because only small segments of the intestine were involved.

Reiner, Schlesinger, and Miller[10] were of the opinion that aureomycin and

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chloramphenicol were responsible for the bowel lesions in 7 of their patients because they could find no other significant etiologic agent.

In a study by Pettet and associates[8] of 107 patients with postoperative pseudomembranous enterocolitis no statistically significant increase in this pathologic entity was noted during the years 1947 to 1952 as compared to the years 1925 to 1946. In 87 of the 94 patients with extensive disease the peritoneal cavity had been opened. In 77 patients the gastrointestinal tract had been directly involved in the surgical procedure. The commonest lesion for which the patients had surgical procedures was carcinoma of the colon (41 of 94 patients). These investigators believed that it was significant that their study showed the incidence of pseudomembranous enterocolitis much higher after operation for carcinoma of the colon than for any other condition. Their patients complained of abdominal pain, distention, nausea, vomiting, diarrhea and shock. Diarrhea occurred in less that half of the patients while shock was a predominant finding during the final few days of life in the majority of patients. Fifty per cent of those patients developing shock died within 12 hours and 70 per cent died within 48 hours. Most of the patients had elevations of temperature to more than 100 F. About 17 per cent developed clinical evidence suggesting cerebral damage. Oliguria was observed in almost all of the patients who lived more than a few hours after the dramatic change in their physical condition. Chemotherapeutic or antibiotic drugs had been administered in 40 of their patients who came to autopsy with the diagnosis of postoperative pseudomembranous enterocolitis.

It is believed that certain antibiotics so alter the intestinal flora that an overgrowth of bacteria, notably staphylococci may achieve pathogenic proportions. Jackson and associates[5] reported diarrhea in 37 to 91 patients treated with terramycin for pneumonia. Stool cultures were made in 18 of these patients and in 12 staphylococcus aureus, resistant to penicillin, terramycin and aureomycin, was found as the only/or predominant organism. Some patients with diarrhea due to the staphylococcus organisms have been markedly benefited when erythromycin was substituted for terramycin, penicillin, or aureomycin. In some patients, the discontinuance of all drugs resulted in improvement. This choleriform syndrome is ascribed to an *entero-toxemia* produced by an overgrowth of resistant staphylococci after suppression of the normal flora of the intestinal tract by potent antibiotics. Finland and Haight,[2] in a study of antibiotic resistant staphylococci at the Boston City Hospital, included 33 strains that were the only or predominant organisms in patients with severe diarrhea, most of them complicating terramycin therapy.

Penner and Bernheim[7] believed that shock was the one important common condition present in all of their patients. Reiner and associates[10] suggested that aureomycin and chloramphenicol were responsible for the production of the lesions. Pettet[8] emphasized the importance of carcinoma of the colon with obstruction and dilatation. In their patients, the pseudomembranous lesions were found proximal to the site of the operative procedure. Prohaska and co-workers[9] used adrenocorticotrophic hormone in 3 patients with postoperative pseudomembranous enterocolitis with prompt and complete recovery. They used intramus-

cular injections of 20 mg. of corticotropin every 6 to 8 hours for 3 to 5 days. Any bacterial disease should have been markedly aggravated by this therapy. They, therefore, do not believe that the disease is an infection, nor do they think it is of endocrine origin. One of the possibilities that they are exploring is that the tissue necrosis results from an antigen-antibody reaction.

Death from postoperative pseudomembranous enterocolitis had been reported prior to the advent of antibiotics, and it would seem, therefore, that its relationship to antibiotics is questionable.[3, 7, 9] It seems reasonable to assume that postoperative pseudomembranous enterocolitis is a different entity from the diarrhea following antibiotic therapy and suppression of the normal intestinal flora. The cause of postoperative pseudomembranous enterocolitis has not been determined, and it has not been determined whether or not antibiotics are contributing factors to this disease.

REPORT OF CASES

Case 1. Mr. T. A., an 81 year old white man first was admitted to Vanderbilt University Hospital on July 4, 1929, with a traumatic stricture of the urethra. He was readmitted on Sept. 16, 1952, with a diagnosis of calculus and diverticulum of the urinary bladder. The third admission was three months later with an additional diagnosis of pericystitis with abscess. The abscess was incised and drained. He was admitted on two other occasions with the same diagnosis. The last admission was on Aug. 26, 1953, because the superpubic abscess had become larger and was more painful. Physical examination was essentially the same as on the previous admission. There was a 10 cm. wide, firm, tender mass in the superpubic region with a draining sinus surrounded by granulation tissue. The leukocyte count was 26,050 per cu. mm., hemoglobin was 9.5 Gm., and non-protein nitrogen 41 mg. per 100 cc. The urine showed 4 plus albuminuria with red and white cells. Culture revealed proteus organisms. On August 27, incision and drainage of the abscess was done, and a biopsy section was taken of the sinus tract. Proteus, E. coli, and paracolon organisms were cultured from the abscess. Biopsy showed metastatic anaplastic adenocarcinoma. He did well in the immediate postoperative period, but on September 1, his temperature rose to 103.8F. and then to 105.8F. on the following day. Rectal examination revealed a large, tender mass above the prostate. He developed apnea, cyanosis, clonic convulsive movements, and became unresponsive. There were rales in the right lung, and a chest roentgenogram showed some infiltrative changes. Penicillin and streptomycin were discontinued, and he was placed on chloromycetin 250 mg. every six hours. This drug later was discontinued, and again he was placed on streptomycin and penicillin. He received oxygen and other supportive therapy. His temperature improved somewhat, and he became responsive. On Sept. 12, 1953, his temperature again rose to 103.4F. Two days later, he suddenly stopped breathing and could not be resuscitated.

There was no record of diarrhea during his illness. At autopsy he was found to have an extensive anaplastic carcinoma of the bladder with widespread metastases, and lesions of pseudomembranous enterocolitis of the rectosigmoid colon. The latter appeared as islands of fibropurulent exudate attached to the superficial portion of the rectal mucosa. Microscopically, there was superficial necrosis and all layers of the intestine contained many inflammatory cells. Cultures of the feces were not made. The final diagnoses were: 1. Extensive bladder carcinoma with lower pelvis and abdominal wall invasion, retroperitoneal and mesenteric lymph node metastasis and hepatic metastasis, 2. Pericystic and superpubic abscess, 3. Multiple bladder diverticula with calculi, 4. Bilateral pyonephrosis and pyoureter, 5. Right and left ventricular hypertrophy, 6. Early calcific aortic stenosis, 7. Pulmonary emphysema, 8. Multiple splenic infarcts, 9. Benign prostatic hypertrophy, 10. Cholelithiasis, and 11. Pseudomembranous enterocolitis of the rectosigmoid colon.

Case 2. Mr. J. W., a 62 year old Negro man first was admitted to Nashville General Hospital, on Aug. 31, 1953, with the history of a lession on the posterior surface of the tongue for two months. Biopsy showed squamous cell carcinoma of the tongue, grade II. The remainder of the physical examination was not remarkable except for enlarged regional nodes in the left neck. On Sept. 4, a hemiglossectomy, hemimandibulectomy, radical neck dissection and tracheostomy were done. Microscopic section of the neck nodes showed metastatic carcinoma. His postoperative course was uneventful and he was discharged on September 18.

On Oct. 6, 1953, he was readmitted with a diagnosis of recurrent carcinoma of the tongue. Radon seeds were implanted about the lesion and 3600 roentgen therapy units were given through two portals to the neck. He was followed at regular intervals after discharge from the hospital. The ulceration in the posterior pharynx was slow to heal; however, three biopsies at monthly intervals failed to show any remaining carcinoma of the tongue or pharynx.

Two months prior to the last hospital admission he began to have dysphagia, which gradually progressed to the point where he could swallow only liquids. An esophagogram showed an obstructing lesion in the midesophagus just below the corina. On esophagoscopy the lesion was encountered 25 cm. from the lower gum margin. Biopsy showed squamous cell carcinoma. He was admitted to the hospital and given preoperative high caloric, high vitamin feedings through a polyethylene tube. All blood chemistries were normal and no evidence of distant metastasis could be found.

On Aug. 10, 1954, esophagectomy was done by two operative teams. The right chest was opened through an anterior incision in the third interspace. The azygos vein was divided and the lung was retracted anteriorly. The esophagus was dissected from the thoracic inlet to the esophageal hiatus. The tumor was found to be adhered to the hilar structures and could not be completely removed. Enlarged lymph nodes containing cancer were noted about the cardia of the stomach. The abdomen was opened through a midline upper abdominal incision and the stomach freed in such a manner as to retain the right gastric and right gastroepiploic vessels. The stomach was brought up into the chest and was found to extend easily and without tension to the apex of the right pleural cavity. The esophagus was divided at the cardia and the stomach end closed. The esophagus again was transected about 5 cm. proximal to the tumor. An esophagogastric anastomosis was made by means of a two layer interrupted cotton technic. A large chest tube was brought out through the right sixth interspace and a Levine tube was passed into the stomach. Both wounds were closed in anatomic layers with interrupted cotton sutures. At the end of the procedure his condition was considered good.

Following the operation he was given penicillin, streptomycin and terramycin. His immediate postoperative course was good. The highest febrile response was 100 degrees. The chest tube was removed on the fourth postoperative day, and at that time the lungs were clear. The Levine tube was removed on the fifth postoperative day and he was started on water by mouth. On the sixth postoperative day at 2:00 p.m. he developed an explosive, voluminous bloody diarrhea and went into shock (blood pressure 80/60). Three hours later he was found to have a complete right hemiplegia and was moribund. During this series of events examination of his stool by Gram stain showed only one organism, a bipolar sporeforming, Gram positive rod which unfortunately never was identified. At 7:00 p.m., five hours after the onset of diarrhea, he was given 100 mg. of cortisone intramuscularly. Within 30 minutes his blood pressure was back to normal. Three hours later the diarrhea had stopped. He was given another 100 mg. of cortisone and this was repeated every 8 hours thereafter. The following day he received 1500 units of whole blood. Following this dramatic response he became greatly improved and was able to indicate his wants. Many rales and ronchi were noted in the lungs and he could not cough. It now was realized that a simple neuroanatomic feature had been overlooked. Because of the previous hemiglossectomy, mandibulectomy and radical neck dissection on the left and the com-

plete hemiplegia on the right, he no longer had sufficient innervation of the muscles of deglutition and respiration to prevent aspiration of oropharyngeal secretions. A tracheostomy was done with excellent improvement. On the seventh postoperative day his chest bandage was found to be saturated with serosanguinous drainage from the stab wound from which the chest tube had been removed. Another chest tube was inserted and connected to an underwater trap. Prior to starting the cortisone therapy, the eosinophile count was 68.75 per cu. mm. By the following day it had dropped to 0 and remained that way as long as he was on cortisone. Repeated stool cultures showed no pathogenic organisms. During the ensuing weeks he improved gradually but still was unable to swallow. A moderately good nutritional state was maintained with tube feedings. His electrolytes were supplemented as indicated by parenteral fluids. He subsequently developed an empyema of the right pleural cavity. The cortisone was decreased gradually from the thirteenth to the fortieth postoperative day when ACTH was started. He was given chloromycetin to combat the empyema.

On the twenty-first postoperative day he developed thrombophlebitis of the deep vein of the left lower extremity and heparin therapy was started. The thrombophlebitis subsided and did not recur. Despite considerable efforts to get him to gain weight he continued to lose and his course was progressively downhill. The most disturbing and unremedial complication was the right hemiphlegia and the continued aspiration of oral secretion. At no time was there any sign of leakage from the anastomosis. His lungs finally filled up and he died on the fifty-sixth postoperative day. He was found to have a diffuse purulent peritonitis and only a small empyema tract in the chest. The lungs were completely filled. The site of the anastomosis was intact.

COMMENT

The patient in case 1 had the only proved case of postoperative pseudomembranous enterocolitis at Vanderbilt University Hospital in 3500 autopsies from the year 1925 to 1954.

The patient in case 2 had extensive surgery, and like most of the reported cases of pseudomembranous enterocolitis this patient had a successful immediate recovery and was progressing well until the sixth postoperative day when, without warning, he developed severe diarrhea with blood, abdominal pain, distention and shock. Within three hours he became paralyzed on the right side. A diagnosis of pseudomembranous enterocolitis was made. He was given an intramuscular injection of 100 mg. of contisone with dramatic improvement. The blood pressure returned to normal within 30 minutes, and the voluminous bloody diarrhea stopped three hours later. The temperature returned to normal, and there was no further abdominal pain. This patient was receiving intravenous terramycin, penicillin, and streptomycin at the time of this complication. Stool examination showed a bipolar, sporeforming Gram positive rod which never was fully identified. Unfortunately, because of previous radical surgery on the left side of the tongue and neck for carcinoma and paralysis of the right side of the tongue following esophagectomy for carcinoma, he was unable to swallow. Because of lung complications a tracheostomy was necessary. He developed pleural emphyema on the right side, decubitus ulcers over the bony projections of the right foot and pelvis, bladder infection, and finally died 56 days after operation. It is interesting to speculate why he developed a diffuse purulent peritonitis which was not suspected prior to death.

SUMMARY

Postoperative pseudomembranous enterocolitis is discussed and 2 cases presented in detail. Cortisone produced a dramatic response in 1 patient even though he finally died from other complications of his primary disease and surgery.

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DIAGNOSIS AND MANAGEMENT OF BRAIN INJURIES

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At the Second Annual Assembly of the Southeastern Surgical Congress, March 9, 1931, my father spoke on the "Diagnosis and Management of Brain Injuries." I have been honored on this, the Twenty-fifth Annual Assembly of the Southeastern Surgical Congress, by being asked to discuss the same title.

Except in lay and legal circles, fractures of the skull have progressively decreased in importance, whereas realization of the damage to the underlying brain has become increasingly important. Linear fractures of the convexity, except those crossing major vessels, are truly of no clinical import. On the other hand, linear fractures of the base, and localized compound depressed skull fractures with brain laceration can be dangerous. Penetrating brain wounds cannot occur without a break in the bony shell. Basilar skull fractures in particular can be recognized, usually by bleeding from the nose, mouth or ears. Brain exuding from a scalp wound gives immediate affirmation of a compound skull fracture with brain laceration. The majority of these fractures can be suspected without moving the patient about in the earlier phases of his injury, when further harm might be done. Roentgenograms of the cervical spine should be made first of all in patients who have been or still are unconscious before the head is turned about for roentgenography. Roentgenograms of the skull always should be made as a matter of record before discharge from the hospital.

Many times severe brain damage occurs without bony handicap. The gelatinous brain may be thrown about and cut against the inside of the skull without any break in the bone. The brain stem may be forced against the sharp edge of the tentorium producing irreversible brain stem damage without any bone change. Subdural hematomas, particularly, frequently are seen without bone damage.

INITIAL HISTORY AND EXAMINATION

The mechanism of injury is of particular value. One is much more apt to see subdural hemorrhages with polar injuries, such as happens to the lady who strikes the back of her head on an open cabinet door, or to the man who bumps his forehead on the low place while going down the cellar stairs. Direct blows to the temple are most apt to result in middle meningeal hemorrhage, as was afflicted by David on Goliath. Linear temporal fractures make one suspicious of the possibility of middle meningeal hemorrhage. Parietal direct injuries are apt to cause intracortical bleeding. The relatively slow broad pressure which occurs when a head is squeezed between two large objects, such as a turned-over trailer and the ground, a slowly moving elevator and the side of the elevator shaft, or a trailer and a loading platform, may fracture the skull like an egg shell and still

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not produce cerebral damages, even though cranial nerves are much disturbed. A sharp corner usually produces a depressed fracture, with small localized depressions. The rapid deceleration which occurs in automotive collisions is effective in producing severe brain lacerations and contusions, many of which are fatal.

The initial examination is of particular value to establish a landmark for further change and to determine other injuries. *Shock* is uncommon in uncomplicated head injuries, usually being due to severe bleeding around extremity fractures, intra-abdominal bleeding, or massive chest injuries. A shock-like state is seen only in brain stem lacerations and medullary decompensation. These usually can be recognized by extensor spasms of the extremities, a rapid feeble pulse, stertorous respirations and subnormal, then rapidly climbing temperatures, and usually fairly rapid exitus. Frequent blood pressure, pulse and respiratory readings should be recorded. Increase in blood pressure and pulse pressure, slowing of pulse and respirations are the classical signs of beginning danger. The person with a head injury does not always show such signs, or may show them in only one sphere of such observations. When the medulla gets less blood it calls on the heart to force more blood into the head, hence the shifting signs. *Rectal temperature* should be determined every hour, as it gives further indication of general bodily difficulties, as well as modifying the other vital signs. A careful look at the eyes may tell the whole story. The size and equality or inequality of *pupils* and their ability to react to light should be recorded early, as shifting of these signs is of particular value in recognizing middle meningeal hemorrhage, the prime neurosurgical emergency. Early examination of fundi gives a base line. Papilledema takes at least six hours to develop, but subhyaloid hemorrhages occur rapidly with severely increased intracranial pressure. The eyeballs frequently are divergent due to unconsciousness. Conjugate deviation of the eyes should be looked for. When this occurs the eyes are turned away from irritative lesions but toward paralytic lesions. The symmetry of facial movements, movements of arms and legs, as well as the tendon reflexes, abdominal reflexes and Babinski's signs should be recorded early. As pathologic signs increase, so should the surgeon's alarm.

The state of consciousness ordinarily gives a fair indication as to the degree of brain damage which has occurred. The patient who is in shock but can tell you his name and what happened to him ordinarily shows mainly associated injury, while the comatose or severely agitated patient who obeys commands poorly and can tell you nothing about himself should be watched closely for head injury complications. The man who at first has responded well and later does not is in trouble. The consciousness therefore must be checked at regular intervals.

MANAGEMENT

I. Patients With Rapidly Increasing Intracranial Pressure. I-A. *Massive brain injury with evidence of rapid exhaustion of the medullary centers.* These patients, the decerebrates, usually die no matter what treatment is employed. Provided the brain stem laceration is not too severe, and they are left

alone except for life-saving measures, occasionally one can salvage patients with stem signs but without true increase in pressure. Dramatic surgical efforts in such patients will only decrease their chance for survival. Early in World War II the wounded with decerebration all died if operated upon, hence later they were set aside. Some carefully nursed patients in civil life have survived with minimal signs.

I-B. *Patients with rapidly increasing intracranial pressure well within the period of medullary compensation.* Rarely do we decompress such patients now unless a diagnosis of localized clot is suspected. A fair number will be lost even with surgical efforts but some can be salvaged. Improved airway is of considerable help in reducing anoxic aggravations for these patients.

II. Patients Presenting Evidence of Middle Meningeal Hemorrhage. These patients, who can be recognized by unilaterally dilated pupil, progressive face, arm then leg convulsions, or paralysis and deterioration of consciousness, should, when suspected, be operated upon immediately and preferably before their consciousness begins to fade. A neurosurgeon may not be near, so the general surgeon must know how to care for these patients. A young surgeon called from a small town in Alabama saying he had a patient who had recently been injured in an automobile accident with deepening coma, large left pupil, marked right facial weakness and lesser arm and leg weakness. Could she be sent up, or would the essayist come down? Obviously, there was not enough time for either. He had no trephine, perforator or burr, so the use of a mallet and chisel to make a hole was suggested. A rongeur was available to enlarge the opening and bone wax to stop bone bleeding. There was no brain suction tip, so one with a finger hole was blown from glass tubing. There being no Bovie he heated forceps in a flame. In that way he saved her life.

III. Simple or Compound Depressed Fractures of the Skull. If there is no evidence of brain damage, a simple depressed fracture of the skull is not an emergency. With localizing increasing neurologic signs, however, early surgery is indicated. Compound depressed skull fractures require thorough debridement, including removal of damaged underlying brain and control of cortical bleeding, as soon as the injured are able to tolerate such efforts. Nowadays surgery can be postponed for many hours with safety and an ample supply of blood helps to prepare patients more quickly for operative treatment. Patients who have massive frontal injuries with compound fractures into air-containing sinuses should have damaged brain tissue removed, cortical bleeding controlled, the dura closed in water-tight fashion, all loose bone radically removed, and sinus membranes stripped as soon as it is safe to operate. Meningitis thus is avoided.

IV. Patients Presenting Definite Evidence of Brain Injury but With No Findings Suggestive of Acutely Increasing Intracranial Pressure. In this group our ideas have shifted considerably over the past 24 years. Hypertonic fluids rarely are used in such patients any more, and ordinarily they are given adequate fluid intake. If one is going to use dehydration, first hypertonic sugar solution should be given intravenously, then within an hour, a magnesium sulfate rectal injection should be given which is drained off at the end of another

hour or so. This is much more effective than either measure alone inasmuch as the fluid is mobilized from the tissues and then removed from the body. Nursing care as outlined below is of major importance in this group.

V. Patients in Whom the Roentgenogram Shows Presence of Linear Fractures but Repeated Examinations Fail to Give Evidence of Intracranial Damage. Here also our ideas over the past 24 years have changed. Those who have no complaints are encouraged to get up out of bed as soon as the spirit moves, and after repeated examinations have shown nothing, are encouraged to return to normal life as rapidly as possible. Most infants and small children will react to any injury with vomiting. This is a shock-like reaction. The safest way to be sure about these patients is to have them where they can be awakened at least hourly. It is not necessary to keep them awake, a mistaken layman's concept.

VI. Rare Types of Injuries Not Falling into Any of the Above Groups. A. *Injuries of the vein of Galen or the tributary veins of Galen.* The only thing one can do for these patients is to nurse them and hope that recovery might occur. B. *Acute and chronic subdural hematoma.* The acute collection of blood in the subdural space originates from a torn blood vessel under the point of maximal force. This is a moderately acute emergency, and with progressive diminution in consciousness a trephine should be made and decompression followed at the point of maximal force. The chronic entity has come to be recognized more frequently over the past 24 years. Due to the usual confusion of neurologic signs in these patients, bilateral trephine openings should be made in those patients with a story of a polar head injury, with or without loss of consciousness, but with persistent headache and at times some neurologic signs. Spinal fluid protein almost invariably is elevated in these patients. C. *Isolated subcortical hematomas.* These are particularly prone to occur in the arm area of the frontal lobe and usually underlie the point of maximal force at the time of head injury. Once such clots are recognized they should be evacuated. D. *Pneumocephalus and cerebrospinal rhinorrhea.* Many times patients with basilar skull fractures will stop leaking spinal fluid if positioning is maintained for one to two weeks with the draining ear or the nose upright, and the head held in one place with sand bags. Should they continue to leak, dural defects may have to be repaired with suturing or fascial transplants along the posterior wall of the frontal sinus or in the region of the cribriform plate. Rarely is it necessary to repair any otitic cerebrospinal leaks. Nowadays with chemotherapy and antibiotics the risk of meningitis in these patients is much less and treatment for meningitis is more effective. Sulfa drugs, being excreted quantitatively into the spinal fluid, are most effective against the usual organisms of the upper respiratory tract. Those patients who are vomiting may be given penicillin, streptomycin or other antibiotics, although none of these crosses the blood-brain barrier as effectively as do Gantrisin® and sulfadiazine.

General Treatment

Airway. The maintenance of a clear airway is one of the major improvements in treatment for such patients. With a powerful suction machine, a no. 20

French catheter with three holes near the end can be passed through the nose without suction, then advanced into the trachea and aspirated for not over 15 seconds at a time with considerable help in removal of secretions. Bronchoscopy or even tracheotomy may be necessary at times.

Shock. Recognition of associated injuries and treatment of these is most important. With a clear chest and abdomen and no injuries to any extremity, shock is rare. Occasionally blood replacement may be needed for scalp bleeding alone. We must replace blood lost to the circulation from bleeding around fracture sites, into the retroperitoneal space, and into the chest and abdomen. Plasma and plasma expanders may be of some help in immediate life-saving measures. Carefully matched blood is much more effective than plasma and plasma expanders in preparing a patient for surgical treatment. The use of external heat has become less popular over the past quarter century since this taxes a limited blood volume. Caffeine sodium benzoate still is of value in these patients.

Hyperpyrexia. Slight rises of temperature can be controlled by the use of aspirin by mouth or by rectum and by avoiding a hot room and excessive covers. Most patients who have head injuries do not sweat, hence cold water or alcohol sponges may be of help by evaporation. If the skin is already cold, further cooling is useless. Friction rubs with a Turkish towel thus are needed to bring the blood into the skin, where one then can cool it. Sedatives as discussed below are of value in reducing the degree of body heat produced. At times one must even resort to cold colonic irrigations. Spinal puncture is of value in the patient with fever due to subarachnoid bleeding. Routine spinal puncture early, before the patient has stabilized, and also in the presence of increased pressure, carries a fairly high risk. Spinal puncture should be done on all patients who have head injuries before discharge for accurate record.

Listlessness and irritability. One frequently sees these signs in head injury patients. Opiates, other than codeine, carry four main dangers: They constrict the pupils, preventing early recognition of pupillary difference; they cut down the chance of observing what is going on in the patient's fundi; they change the patient's state of consciousness; and, of major importance, they depress his respirations. These include morphine, pantopon, dilaudid and demerol. The use of sodium phenobarbital in 2 to 5 grain doses as often as every four hours, of APC compounds with codeine for pain, and the use of chloral hydrate, bromides, or paraldehyde often are of considerable value in such patients. It is well to remember that the agitated patient becomes much more active when he has a full bladder, hence an indwelling catheter may be needed. Physical restraint of these patients who otherwise might pull out their catheters, only increases their agitation and therefore should be avoided. Frequently the patient wants to sit up to breathe better or to void. If these patients are allowed some freedom instead of being held on the bed, the restlessness frequently will subside. The use of *boxing gloves* made of bandages will eliminate the contact of thumb and finger and thereby many times leave tubes undisturbed, and still allow the patient to scratch where it itches. *Convulsions* require delicate handling. In the absence of dilated pupil and increasing drowsiness sodium phenobarbital intramuscularly and

Pentothal® or Surital® intravenously frequently can prevent the patient's damaging himself.

General care of such patients, with tube feeding after vomiting has ceased, helps in approaching physiologic conditions. The Levine tube also gives a ready route for medication.

SUMMARY

These patients actually fall into three categories. Around 20 to 30 per cent of them, the Lord will take care of, and He usually does so rather promptly in spite of everyone's best efforts. The second group is cared for by the nurses and consists of 50 to 60 per cent of such cases. The surgeon here must direct the patient's care. Finally, about 5 to 10 per cent of these people will require some surgical operation to attack localized clots. The surgeon still must assume the primary responsibility in all patients in order to be sure that he can recognize the small percentage that requires more active intervention. He also must be well versed in the nursing care of the patient in order that he may instruct and supervise the available help in their efforts. Middle meningeal hemorrhages particularly should be recognized as soon as advancing unilateral neurologic signs are noted. Although the mortality rate in this group of patients should be nil, it still hovers in the range of 20 to 25 per cent in any large series of head injuries, since operation frequently is postponed until irreversible damage has occurred. It is better to find the clot in the operating room than in the morgue.

A CASE OF MULTIPLE LEIOMYOMAS OF THE ESOPHAGUS*

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Benign tumors of the esophagus are about 1 per cent as common as carcinoma of the esophagus.[1, 3, 4] Less than 200 definitely proved cases have been recorded in the entire world literature.[8] Seventy-five have been leiomyomas which are solid intramural tumors and usually are in the form of solitary lesions.[2]

It is important to call attention to these tumors, for until recently they have not been properly recognized or treated. At first they were discovered only at autopsy. One-third of the patients with polypoid tunors, and one-sixth of those with solid intramural tumors died as a direct or indirect result of their lesions.[7]

At the time we operated upon our patient only 35 leiomyomas and 30 other benign tumors of the esophagus had been removed surgically. With an increased index of suspicion and with the aid of modern radiology, esophagoscopy, and especially negligible-risk thoracotomy, these lesions should be discovered early and resected. The patient's general condition then is good, there is a good chance for complete resection, and less possibility of malignant degeneration.[6] Thus the patient is relieved of his symptoms sooner and a permanent cure is achieved.

CASE REPORT

A 31 year old Negro man was admitted to Lincoln Hospital on Oct. 4, 1953, complaining of generalized, intermittent, crampy abdominal pain. This started a few hours after a large meal with excess alcoholic intake. There was no localization of the pain. He had not vomited. There were no other significant symptoms. Over a period of five years the patient had had several episodes of indigestion characterized by heartburn and vague epigastric pain, usually relieved by alkali. There had been no difficulty in swallowing. In 1939 and again in 1951 he had had moderately severe pneumonia. Otherwise there was no history of respiratory symptoms. Systemic review was not remarkable. The patient was well developed and well nourished. Physical examination was negative except for moderate generalized abdominal tenderness. The complete blood count, urinalysis, and serology all were normal. The initial impression was alcoholic gastritis. The patient made a satisfactory improvement on symptomatic, antiulcer type treatment.

Radiologic examination of the upper alimentary tract on October 7, showed no abnormality of the stomach or duodenum, but fluoroscopy of the chest showed "a mediastinal mass just below the level of the aortic knob which displaces the esophagus posteriorly and to the right." Barium swallow showed the esophagus displaced sharply backward by the mass. The radiologists suggested that the mass could be a primary or metastatic mediastinal tumor, or possibly a saccular aneurysm on the posterior wall of the aorta. The impression was that it was probably extra-esophageal, and certainly extra-mucosal. Esophagoscopy thus was thought to be of no value. The patient had continued to improve, was ambulatory, and by October 18 was on a regular diet. He was in excellent general health

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FIG. 1 a

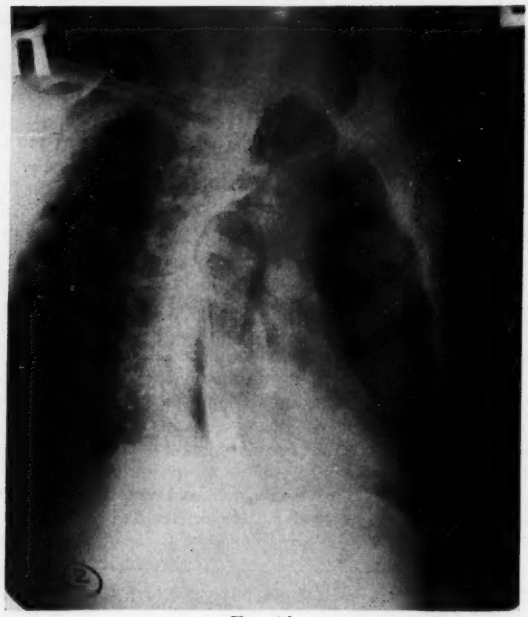

FIG. 1 b

FIG. 1 (a and b). Preoperative roentgenograms showing mass outlined by barium swallow

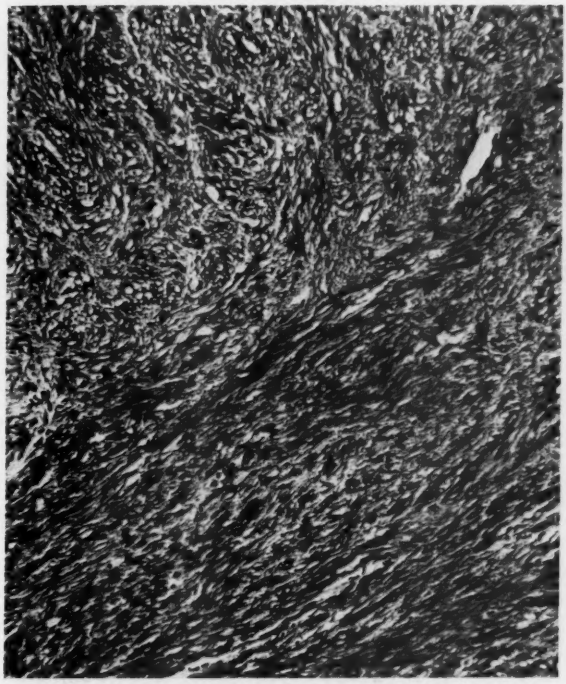

FIG. 2. Photomicrograph of resected specimen showing typical leiomyoma. High power

and required very little preoperative preparation. Thoracotomy was done both for diagnosis and treatment.

On October 22 under ether anesthesia a thoracotomy was done through the bed of the resected right fifth rib. Three separate tumor masses, 4 cm., 1.5 cm., and 1 cm. in diameter were enucleated from the wall of the esophagus at the junction of the upper and middle thirds under the arch of the azygos vein. It was necessary to divide the vein in order to approach the tumors. The largest of the masses was so adherent that the mucosa could not be separated and the lumen was penetrated at one point. This was quickly closed with fine chromic catgut, which also was used for loose closure of the muscle fibers. Frozen section diagnosis of leiomyoma confirmed the gross impression.

The patient did well postoperatively. He was fed by nasogastric tube from the second to the eighth day, when he was started on liquids by mouth. The tube then was removed. He was taking solid food without difficulty by the tenth postoperative day.

Terramycin was administered for one week. No significant reaction developed from operative penetration into the esophageal lumen. Drainage from the pleural cavity amounted to 500 cc. and was clear after the first day. Chest roentgenograms showed a slight pleural thickening, but the mediastinum was of normal configuration and in normal position, and the lungs were well expanded.

Eighteen months following the operation the patient was completely asymptomatic. A chest roentgenogram was not remarkable. Barium swallow showed slight widening of the esophagus at the site of the resection, but there was no delay in the passage of the barium into the stomach. The patient was on no medication or limitation of diet.

DISCUSSION

Patients from 19 months to 66 years of age have been successfully operated upon for benign tumors of the esophagus. Males outnumber females. The majority of leiomyomas occur in patients at about 40 years of age. Our patient was a 31 year old man.

We have been able to find only 1 other recorded case of leiomyoma of the esophagus in a Negro person. This especially is interesting in that leiomyoma of the uterus is so common in this race.

Multiple tumors occur in less than 25 per cent of the reported cases of leiomyoma. This patient had three separate nodules. In spite of this multiplicity he had no dysphagia or other localizing symptoms. Dysphagia has occurred in about one-half of the surgically removed leiomyomas, and in one-third of all cases reported.

It is not unusual that this lesion first was discovered during a routine fluoroscopy of the chest,[5] but subsequent radiologic study certainly was misleading in that the lesion appeared to be completely extrinsic to the esophagus. This apparently was due to the confusion of superimposed shadows at the high level at which the tumor occurred.[7]

SUMMARY AND CONCLUSIONS

A case of multiple leiomyomas of the esophagus has been presented.

Although symptoms tend to be vague in such lesions, with proper study they ultimately should lead to early diagnosis.

The lesion often can be demonstrated preoperatively by careful radiologic examination. Esophagoscopy, physical examination, and laboratory studies are of secondary value in the consideration of solid intramural tumors. Easily available polypoid lesions, however, often may be diagnosed and treated by esophogoscopy.

Ultimate diagnosis and permanent cure require early thoracotomy in most instances. The entire lesion should be resected. Frozen section examination should be made to rule out malignancy in which case more radical surgery may be indicated. The risk is small. Significant complications are rare.

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ANNULAR PANCREAS: REPORT OF A CASE

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Annular pancreas has been described as a congenital anomaly in which a firm ring of grossly recognizable and apparently normal pancreatic tissue partially or completely encircles the descending portion of the duodenum and is confluent with the head of the pancreas. Although thought to be rare, cases in increasing number have been reported during the past decade. In 1953 Castleton and associates[3] reviewed 32 cases in which operation was performed, 75 per cent of which had been published since 1940. A case encountered by us will be reported in the interest of clarifying the clinical findings and the type of operative intervention in those congenital lesions causing symptoms in adulthood.

EMBRYOLOGY

The pancreatic anlages appear during the fourth week of embryologic life as two thickenings of the wall in the caudal portion of the foregut. The dorsal anlage, the larger, develops into the tail, body and ventral portion of the head of the adult pancreas. The ventral anlages, both right and left, show slightly different development. Normally, the right ventral bud is rudimentary and disappears early in embryologic development, whereas the left ventral bud enlarges and rotates with the duodenum, ultimately fusing with the dorsal portion to form a portion of the head and uncinate process of the pancreas. Lecco[7] postulated that adherence of the right ventral bud to its point of origin and its failure to rotate with the duodenum would result in formation of an annular pancreas. Whereas other theories have been advanced, this concept of the embryologic development is most widely accepted.

SYMPTOMS

Annular pancreas is one of the few embryologic lesions of the gastrointestinal tract producing initial symptoms either in the first few days of life or after maturity. Encirclement of the descending portion of the duodenum may produce varying degrees of obstruction. Symptoms of annular pancreas are compatible with intermittent, partial or complete obstruction of the upper gastrointestinal tract.

In those patients in whom the normal band of pancreatic tissue results in nearly complete obstruction at birth, vomiting, loss of weight and meconium stools are noted clinically. However, in the majority of patients symptoms develop after the twentieth year. These vary from mild upper abdominal discomfort, bloating and eructation to severe episodes of protracted nausea, vomiting and epigastric pain. In many of the reported cases of annular pancreas, the duo-

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denal obstruction was minimal and no symptoms were recorded during life, the annular pancreas being noted incidentally at necropsy.

DIAGNOSIS

Whereas roentgenologic study is the primary contributor in substantiating the diagnosis, evaluation of the entire clinical picture is essential. The patient usually is seen because of continued complaints referable to obstruction of the upper gastrointestinal tract or because of symptoms simulating those of duodenal ulcer. Carcinoma of the duodenum, ampulla of Vater, or pancreas may be suspected following roentgenologic examination, because of the deformity in the second portion of the duodenum. However, duodenal obstruction appearing late in life, together with absence of occult blood in the stool, anemia and cachexia, is not compatible with the diagnosis of carcinoma of the duodenum or ampulla of Vater. Clinical differentiation of duodenal atresia and annular pancreas in children is of little significance since surgical intervention is mandatory in both instances. At the time of surgical exposure, care must be taken that nodularity and fibrotic firmness of the tissue do not lead one to the erroneous assumption of malignant growth.

TREATMENT

Development of obstructive symptoms is an indication for surgical intervention. Since approximately one-fourth of the cases are associated with other congenital anomalies[8], thorough exploration at this time is important. Two types of operations have been most widely employed. The method first advocated was direct attack upon the obstructing ring of pancreatic tissue with resection of the annulus followed, if necessary, by a plastic procedure upon the second portion of the duodenum. The second method is a by-pass procedure, such as duodenoduodenostomy, duodenojejunostomy or gastrojejunostomy. Goldyne and Carlson[5] collected 52 cases in 13 of which operation was performed with 4 deaths, or a mortality rate of 31 per cent. Duodenojejunostomy or duodenoduodenostomy has been advocated by some as the treatment of choice because of the reported development of pancreatitis, pancreatic fistula or secondary fibrosis and stricture following resection. In a collected series Gillette and Lynch[4] reported a mortality rate of 6.6 per cent and good results in 86.8 per cent of 15 patients in whom the by-pass procedure was performed in contrast to a mortality rate of 22 per cent and good results in only 11.1 per cent of 9 patients treated by resection. Moreover, in 6 patients, symptoms of obstruction or fistula persisted following resection of the annulus.

CASE REPORT

Mr. R. V. P., aged 47, came to the Ochsner Clinic in May 1953 because of intermittent gastrointestinal discomfort of seven years' duration. Gradual onset of anorexia and excessive salivation were noted during the year before admission, the attacks being short lived but often disturbing his sleep. There was no appreciable loss of weight. The patient had had an attack of Bright's disease at the age of five years.

Physical examination revealed left direct, and right indirect, inguinal hernias and moderate, diffuse enlargement of the prostate. The hematogram, sedimentation rate, blood glucose, glucose tolerance curve, cholesterol, and results of serology, urinalysis, fecal

examination, and gastric analysis were within normal limits. The electrocardiogram revealed strongly suggestive evidence of myocardial disease. Roentgenograms of the chest, intravenous urograms, cholecystogram and barium enema examination showed no abnormalities. Fluoroscopy of the upper gastrointestinal tract revealed slight elongation of the pylorus with minimal irregularity but no evidence of obstruction. The radiologist believed that the deformity was the result of a hypertrophied pyloric muscle. The patient was discharged with instructions to take antispasmodics and follow a bland diet.

The patient returned in June 1954, because the symptoms had increased in severity and frequency during the preceding few months. Results of a general physical examination and laboratory studies were essentially the same as on the previous visit, with the exception of fluoroscopic examination of the upper gastrointestinal tract. The deformity of the pylorus previously visualized was unchanged but there was 40 per cent gastric retention at the end of two hours.

Because of the progressiveness of the symptoms and the gastric retention the abdomen was explored through a transverse subcostal incision. The first portion of the duodenum was approximately twice normal size (fig. 1a) but not inflamed or ulcerated. Near the midportion of the second part of the duodenum, a band of pancreatic tissue extended $\frac{2}{3}$ of the way across the anterior aspect. On mobilization of the second portion of the duodenum, a similar band of tissue arising from the pancreas was found to extend approximately $\frac{3}{4}$ of the way across the posterior aspect. This pancreatic band was 0.5 to 0.75 cm. thick and from 2 to 3 cm. wide. No duct was grossly identifiable. The lumen of the duodenum at this point would not admit the tip of the fifth finger following gastrotomy and indirect exploration. An isoperistaltic, posterior gastrojejunostomy (fig. 1c) was done, using an inner layer of No. 000 atraumatic chromic catgut and an outer layer of interrupted cotton sutures. The wound was closed in layers with quilting cotton sutures throughout.

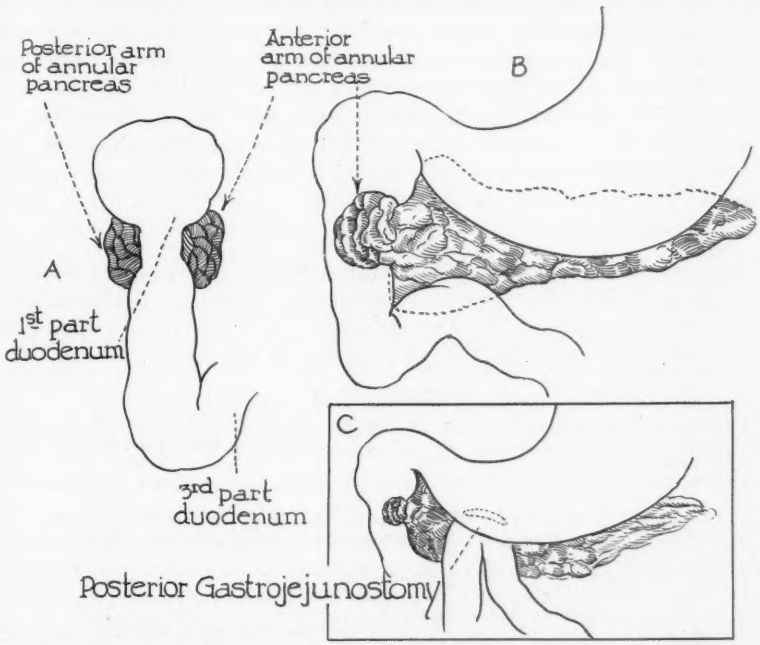

FIG. 1. Diagrammatic sketch of annulus, anterior and right lateral, and posterior gastrojejunostomy.

Recovery was uneventful and the patient was discharged from the hospital on the eighth postoperative day.

COMMENT

Although annular pancreas has come to be a recognized entity, its occurrence still is relatively rare. It is easy to understand the symptoms of duodenal obstruction in the newborn with an annular pancreas, and the necessity for surgical intervention is obvious. It is a little more difficult, however, to explain the development of symptoms in the adult. It has been noted in other patients, as in our own, that symptoms do not appear until later life, at which time the patient experiences vague epigastric discomfort, bloating, mild digestive disturbances, and anorexia, which gradually increase in severity and in duration. Silvis[9] pointed out that in 26 patients who were operated upon, 60 per cent of them in whom the annulus was resected showed changes consistent with chronic inflammation. It has been postulated that the chronic inflammatory changes with accompanying fibrosis produce further narrowing of the duodenum, with the onset of obstructive symptoms. Lehman,[8] Bickford and Williamson,[2] and Anderson[1] all mentioned that following resection of the annulus, there is continued retention of gastric contents. In some instances, retention, noted within a few weeks postoperatively, improves after three or four months.

Gross and Chisholm[6] pointed out that there usually are two arms of pancreatic tissue which fuse, although if a gap is present it usually is anterior, as in the case herein reported. Although there were no gross evidences of pancreatitis and accompanying fibrosis in this instance, and no apparent complete constriction of the duodenum, the lumen of the duodenum on exploration through gastrotomy was considerably smaller than the normal.

Because of the reported high incidence of pancreatic fistula, pancreatitis, and postoperative gastric retention, we consider a by-passing or short circuiting operation, such as duodenojejunostomy or gastrojejunostomy, to be the procedure of choice.

SUMMARY

A case of annular pancreas in a white man treated surgically is reported. The treatment of choice, when obstructive symptoms develop, is duodenojejunostomy or gastrojejunostomy. Since approximately 25 per cent of patients have associated congenital anomalies, thorough exploration is indicated.

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CALCIFIED CYST OF SPLEEN: REPORT OF A CASE*

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Cysts of the spleen are uncommon. Fowler classified cysts of the spleen into: (1) echinococcic, (2) dermoid, and (3) nonparasitic.[6] The nonparasitic type is subdivided into true and pseudocysts. The true cyst, as its name implies, has a cell lining, whereas the pseudocyst does not. It seems that the term, nonparasitic, is the most descriptive name to distinguish a class of cysts of the spleen of varying pathology. It includes the rare calcified cyst which will be discussed later in a case report. It does not include neoplastic cysts, such as hemangiomas and lymphangiomas, which are classified as tumors.

ETIOLOGY AND PATHOGENESIS

Considerable interest is expressed throughout the literature concerning nonparasitic cysts. The fact that so many explanations of the etiology and genesis of these cysts are given is evidence that they are not completely known. Tamaki[17] stated that a review of cases revealed a history of trauma in approximately one-fourth of them. Vascular occlusion with resulting infarction, necrosis, and hemorrhage has been discussed. These have been attributed to embolism, thrombosis, endarteritis, and rupture of one of the smaller arteries of the spleen. Herbut[8] believed that some of these cases are due to abscesses which finally became *burned out*. The majority of cases seen in women are during and after the childbearing period, and for this reason it is speculated that pregnancy may have some influence, since the spleen is more congested during pregnancy. Hormonal influence also has been given as a cause, as also has endometriosis. Tamaki quotes Frank as describing a case of a patient in whom a large cyst was marsupialized and blood was discharged from it at each menstrual period after the operation. Pratt-Thomas and Switzer[12] report a case of cyst of the spleen in sickle cell anemia. This case fortifies the theory of infarction with necrosis inasmuch as sickle cell anemia predisposes to thrombosis in the smaller vessels.

There is confusion about the origin of the lining cells of the true forms of the nonparasitic cysts. There have been several explanations for a mesothelial origin, such as cell rests which in later life begin to grow, abnormal invagination of the peritoneum during growth, and enfolding of the peritoneum of the spleen after a laceration. On the other hand the endothelial origin is explained by ectasia of the sinusoids resulting in *dilatation cysts*, or endothelization of the hemorrhagic area by endothelial cells of the spleen.

Bostick and Lucia[3] stated that the so-called epidermoid (not to be confused with dermoid) cyst of the spleen often is interpreted as implying origin from epithelial precursors and hence is inaccurate. They believed that cells of mesothelial (or endothelial) origin may undergo metaplasia and become stratified, or

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may show stratification in crevices or recesses of the cyst. They further stated that stratification with intercellular bridges and keratinization are in favor of true epithelial origin. Tamaki also believed that a squamous cell lining may result from metaplasia of endothelial cells.

The reason that these cysts, if they are of hemorrhagic origin, do not undergo organization and heal by scar tissue formation, is not explained. Pratt-Thomas and Switzer believed many of these hemorrhagic cysts, or those which are the result of infarction, become transformed into serous ones through absorption of blood and necrotic material with eventual endothelization of the cavity.

Once the pathologic process has started there may be repeated infarctions. The result would be small hemorrhagic infarctions coalescing with the larger cyst. The arteries within the spleen do not anastomose and are so-called *end-arteries* and collateral circulation for an infarcted area cannot be established.

PATHOLOGY

These nonparasitic cysts may be small, only a few centimeters in diameter, or may reach such size as to contain several liters of fluid. The color and consistency of the fluid may vary from a serous or serosanguineous fluid to one which contains blood clots and liquefied splenic tissue. Those of many years duration may develop a calcified wall. Some may develop lining cells and others fail to do so. The true cysts have a tendency to contain a serous or serosanguineous type fluid, whereas the pseudocysts contain hemorrhagic material in a thicker fluid medium.

The microscopic picture is variable. The cyst may or may not be lined with endothelial cells, or may have stratified squamous epithelium. There may be cholesterol crystals about the lining of the cyst. The wall consists of thin fibrous or thickened hyalinized fibrous tissue. Calcified areas in the wall may be present. Leukocytic infiltration also may be present depending upon the extent of inflammatory reaction present.

DIAGNOSIS

The dominant features in diagnosis are upper left abdominal pain and a mass. The differential diagnosis may depend to a large extent upon a thorough history and physical examination. Roentgenograms of the gastrointestinal tract and urograms should be made to rule out pathology of the pancreas, left lobe of the liver, and the left kidney. If the splenic cyst is calcified it can be seen as a smooth round calcified body with a distinct rim about its periphery. If splenomegaly can be detected careful hematologic (including bone marrow) studies should be made. Despite these studies and roentgenograms a diagnosis may not be made except at the time of operation.

CASE REPORT

A. A., a 55 year old white woman, was admitted to the Orangeburg Regional Hospital on Dec. 16, 1954, with the complaint of pain in the epigastrium and upper left abdomen. About four weeks prior to admission she had an acute episode of epigastric pain accompanied by fever and nausea which subsided after several days. She stated that for a period of about six years she had had vague discomfort with a sensation of *fullness* in her upper left abdomen. There was no history of digestive disturbances.

FIG. 1. Abdominal roentgenogram of patient showing a large calcified cyst of spleen. Note the downward displacement of left kidney. The scoliosis is incidental.

There was a history of an automobile accident 10 years ago. She did not recall a direct blow to her abdomen or left flank in the accident, but her body was suddenly and acutely flexed by the immediate deceleration of the automobile. She has had eight full-term pregnancies. Her menopause was in 1946 after a normal menstrual history.

Routine physical examination showed a very obese woman in no apparent distress. The abdominal wall had a thick panniculus which made examination difficult. Deep pressure over the upper left quadrant elicited moderate tenderness. There was a questionable mass in this area. The percussion note over this area and Traube's space was dull. Peristalsis was normal and there was no evidence of a bruit. Except for moderately severe varicose veins of both lower extremities, the remainder of the physical examination was noncontributory.

Routine blood counts, hemoglobin, and urinalysis were normal. Roentgenograms of the abdomen showed a round mass with a calcified wall in the upper left quadrant measuring 15 cm. in diameter situated just beneath the left leaf of the diaphragm (fig. 1). The left kidney was displaced downward. Roentgenograms of the stomach with barium show the stomach displaced medially.

On December 18, she was operated upon. A long upper left transrectus incision was made.

Upon opening the abdomen the spleen was found much enlarged with a mass occupying the upper and medial portions. The gastrosplenic ligament and splenic pedicle were divided between ligatures. The omentum had attached itself to a thinned-out portion of the cyst at the upper medial aspect of the spleen. The attachment was freed by blunt dissection. When this was done the cyst was ruptured and a quantity of light brown semisolid fluid escaped. Cultures of the contents of the cyst were taken. The spleen was adherent to the under surface of the diaphragm. It was freed by dissection with the fingers. The spleen with its mass then was freed and removed. A Penrose drain was inserted down to the space that the spleen had occupied and brought out through a left subcostal stab wound. The abdomen was closed and the patient was returned to her bed. Elastic bandages were applied to her lower extremities.

The gross pathologic report was "a cystic structure measuring 15 by 12 by 15 cm. The spleen extends across the surface of this cystic mass. The cyst proper has a calcified wall and a rough granular tan lining with soft granular and putty-like material of similar color in the cavity." Microscopic examination showed "an irregular thick-walled structure lined by a thick layer of hyalinized material with foci of calcification. This structure contains amorphous material including blood pigment." The pathologic diagnosis was "cyst (subcapsular hematoma, old?) of spleen." A four day culture of the smears taken at the time of operation was reported 100 per cent hemolytic staphylococcus albus.

The patient made an uneventful recovery and was discharged on Jan. 1, 1955. She was followed as an outpatient periodically and was last seen on March 30, 1955. At that time she had no complaints.

COMMENT

The history of trauma given by this patient cannot definitely be ascribed as the etiologic factor for the development of the cyst, but it seems the most reasonable explanation in the absence of other causes. The time interval of 10 years makes it the more feasible. Tamaki reported that 90 per cent of the patients with a history of trauma developed the cysts within 10 years and 50 per cent in 3 years.

It is interesting to note that at the time of operation the omentum had attached itself to a thinned-out area of the cyst. It is likely, according to her history, that she had an acute flare-up of an inflammatory process 4 weeks prior to operation and that the cyst was on the verge of rupturing after becoming secondarily infected as shown by the positive culture of its contents.

The tendency for some of these cysts to calcify cannot be explained. Calcification denotes a process of long duration. Presumably calcium salts are deposited after a hemorrhagic process and the blood is later resorbed.

Despite the variations in the color and consistency of the contents of cysts, the presence or absence of lining cells and calcification, it appears that these nonparasitic cysts are the same entity. The individual variations can be explained by the duration of pathogenic processes, local ability or inability of the spleen to limit the process, or become lined by cells, and pathophysiologic processes of the body influencing the spleen.

Martin[11] and his associates in 1950 found 184 nonparasitic cysts reported. Review of the literature since then and through 1954 places the number at 18 making a total of 202.[1, 2, 4, 5, 7, 9, 10, 12-20] Neidhardt[12] in 1953 recorded the total of calcified cysts as 36 including a case report of his own. It is believed that the

author's case brings the total of nonparasitic cysts of the spleen reported to 202 and calcified splenic cysts to 37.

SUMMARY

The literature on nonparasitic cysts of the spleen has been reviewed with various theoris of etiology and pathogenesis.

The different types of nonparasitic cysts (true, false, calcified, and probably so-called "epidermoid") are believed to be an entity with variations in pathogenic processes.

The author's case brings the total reported nonparasitic splenic cysts to 202 and calcified splenic cysts to 37.

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UNUSUAL METASTASES OF CARCINOMA OF THE PROSTATE, WITH FOUR CASE REPORTS*

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There are here recorded 4 cases, presenting 3 very unusual secondary manifestations of prostatic malignancy. The first case here reported is that of involvement of all the corpora of the penis by direct extension of the prostatic carcinoma. In the second case here reported a rare metastasis of the prostatic neoplasm to the testicle was found. The third unusual complication was that of paraplegia, resulting from pressure on the spinal cord by vertebral metastases of the prostatic carcinoma, as demonstrated in the third and fourth cases here reported.

Involvment of the corpora of the penis by carcinoma of the prostate is infrequent, and thus far only 17 cases have been found recorded in 15 sources.[3-7, 9-14, 17-20] Two of the cases[4, 5] were sarcomas of the prostate with penile metastases, as shown by the microscopic sections.

Martin-Laval[9] referred to Raffin[14] as having reported 2 cases, and Motz and Majewski[10] 1 case, which were similar to his. These two references have not been previously credited, but Martin-Laval's discussion quite clearly refers to their case reports as carcinoma of the prostate with penile metastases.

Priapism usually is the first penile symptom, and is due either to the metastatic infiltration of the corpora, or stasis and thrombosis. The pathway that the cancer cells take from the prostate to the corpora has not been definitely determined, except where direct extension can be shown. Waller and Hellwig[18] favored the theory of a retrograde route of spread by way of the dorsal vein of the penis which connects with the prostatic plexus. In their patient, the metastatic nodule appeared on the glans penis. They did not find any continuity between the prostate and corpora. However, in case 1 here reported, a direct extension was found to have been the route of spread, since continuity between the prostate and the corpora was demonstrated at the time of operation.

Metastasis of prostatic carcinoma to the testicle is a rare occurrence, and has been recorded in but 4 cases. The route of spread was discussed by Bradham[2] and he believed that, in his patient, the testicle had been involved by venous extension, rather than by arterial or lymphatic channels. None of the 4 patients, nor the 1 in the case here reported (case 2), had been diagnosed prior to operation or autopsy.

Paraplegia secondary to vertebral metastatic lesions from carcinoma of the prostate is relatively uncommon. Kawaichi and Rider[8] have found 10 cases recorded in their view of the literature, and added a case of their own. Toulson and Hawkins[16] found 3 cases in 122 patients with carcinoma of the prostate. At the Lake City Veterans Administration Hospital the incidence from 1948 through 1953 was 2 (case 3 and case 4) in 182 patients with prostatic carcinoma.

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CASE REPORTS

Case 1. C. R. N., a 77 year old white man, was admitted to the Lake City Veterans Administration Hospital on April 7, 1953 complaining of urgency, nocturia and hourly frequency. His urinary symptoms began in December 1952, following *intestinal flu*.

He was a well developed and nourished senile white man who appeared chronically ill. On admission, the urinary bladder was found to be distended almost to the xiphoid, and catheterization yielded over 3000 ml. of urine. The catheter was left indwelling. The prostate was enlarged 1 plus, indurated and extended laterally to the right. There were no other significant physical findings except enlargement of both breasts.

On admission, urinalysis showed a specific gravity of 1.007, albumin 1 plus, and many erythrocytes per high power field. Blood serologic test for syphilis was negative. The complete blood count was within normal limits. Serum acid phosphatase was 1 Gutman unit and the alkaline phosphatase was 13 King-Armstrong units. Serum chloride level was 610 mg. per cent, and the non-protein nitrogen was 70 mg. per cent. Roentgenograms of the chest, lumbosacral spine and pelvis failed to show any cancer metastases. Repeat films taken two months later still were negative for metastases. An electrocardiogram taken on April 14, 1953 was within normal limits.

Frequent laboratory studies were made. The non-protein nitrogen level gradually fell, and by June 5, it was 21 mg. per cent. Several blood counts and serum potassium levels were within normal limits. Serum acid phosphatase was 4 Gutman units on May 19, and 1 on May 26.

Clinically, the patient became mentally clear, cheerful and ambulant on treatment with parenteral fluids, urinary antiseptics and indwelling urethral catheter drainage. On April 23, a needle biopsy of the prostate was made, and it confirmed, histologically, the clinical diagnosis of carcinoma of the prostate. Stilbestrol, 25 mg. in oil, was given daily intramuscularly beginning on April 23. Oral stilbestrol was omitted, because of nausea and vomiting, until May 29, when 25 mg. was given four times daily until operation on June 8.

After a few weeks of hospitalization, the patient noticed slowly increasing penile discomfort. At first, this was believed to be due to the presence of the indwelling catheter. Examination of the penis showed a very tender, indurated and thickened proximal half with involvement of all corpora. The probability of metastatic involvement of the penis was confirmed by a needle biopsy of the left corpus cavernosum, done on May 29. The microscopic picture (fig. 1) showed the same anaplastic, fibrosing carcinoma as the needle biopsy of the prostate.

The penile metastases evidently had continued to progress in spite of the 4 weeks of stilbestrol therapy. Because of the extreme discomfort, amputation of the penis and bilateral orchiectomy were proposed. The absence of demonstrable metastases in the chest, spine, or bony pelvis supported the decision for operation. A total amputation of the penis, cystotomy, and bilateral orchiectomy were done on June 8, 1953, through a combined scrotal and retropubic approach. There was far advanced pelvic extension, and by the time the hopelessness of the situation was discovered, the author was committed to completing the amputation.

Grossly the penis was enlarged and much indurated in the proximal two-thirds. There was some degree of firmness in the distal third, and all the corpora were found to be involved on cross-section (fig. 2). Microscopically, there was a diffuse anaplastic fibrosing adenocarcinoma involving the 3 corpora. It was growing in the form of small, poorly developed acini in the prostate, while in the corpora, there was better differentiation into acinar groups with secondary acinus formation.

During the operation, and in the immediate postoperative period, the patient was supported with blood transfusions, intravenous fluids, Levophed and oxygen. The postoperative course was stormy, and in spite of all possible supportive efforts, the patient died on the ninth postoperative day. For 24 hours prior to his death, there was almost complete suppression of urine and both lower extremities were edematous, cool, and somewhat cyanotic. Autopsy permission was not granted.

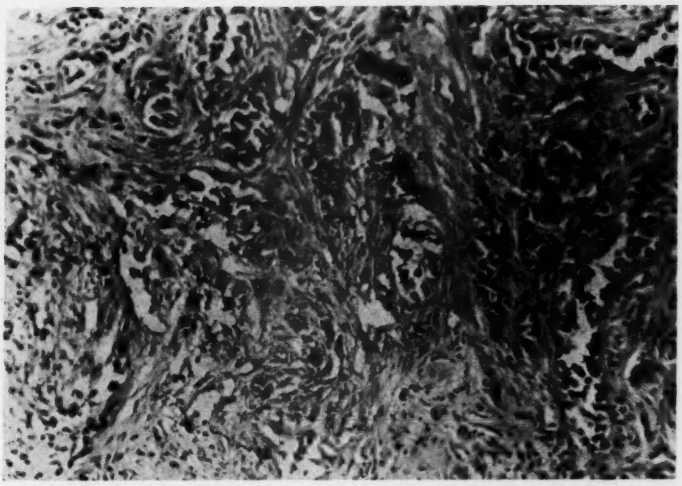

FIG. 1. Needle biopsy of the left corpus cavernosum, showing the extension from carcinoma of the prostate in case 1.

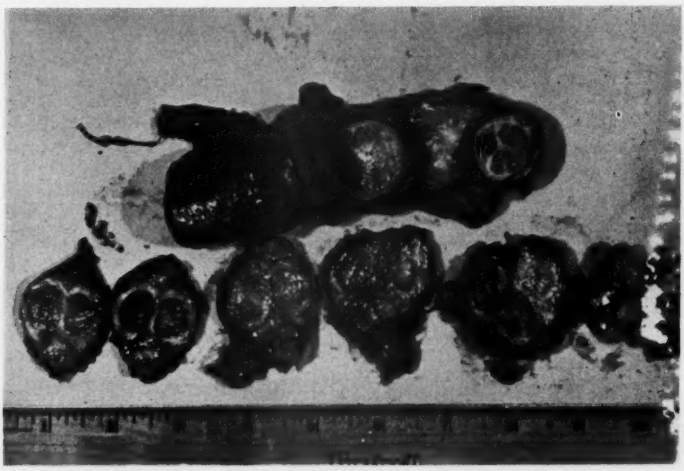

FIG. 2. Series of cross sections through the penis, demonstrating involvement of all three corpora, in case 1.

Case 2. W. J., was a 60 year old Negro man who was admitted to the Lake City Veterans Administration Hospital on April 14, 1951 complaining of hesitancy, dysuria, urgency, nocturia, and a weak dribbling stream. He was ambulant, well developed and well nourished. The only important findings were an enlarged, tender, firm left epididymis, and a 3 plus enlargement of the prostate. Panendoscopic examination showed a coarse trabeculation of the bladder with a 3 plus median and lateral lobe hypertrophy.

Complete blood count, non-protein nitrogen, urea nitrogen, serologic test for syphilis, electrocardiogram, and chest roentgenogram all were within normal limits. The excretory

urograms showed a normal upper urinary tract, and an extrinsic mass of enlarged prostate at the vesical outlet, confirmed by a double contrast cystogram. Serum acid phosphatase was 0.65 Gutman units. Urinalysis showed a 2 plus albumin reaction, with 15 to 20 erythrocytes and 8 to 10 leukocytes per high power field.

He was kept on continuous catheter drainage, and given gantrisin with antibiotics until a retropubic prostatectomy was done. The procedure was quite difficult, resulting in removal of the entire left half of the gland, and enucleation of tissue on the right. The postoperative course was smooth. He was supported with intravenous fluids, antibiotics and gantrisin. After removal of the urethral catheter, he voided freely, and the incision healed rapidly. He was given a 30 day leave, and on his return, estrogenic therapy was initiated because the tissue report was that of an infiltrating adenocarcinoma. Stilbestrol, 25 mg., was given three times a day orally, and 10 mg. intramuscularly daily. A bilateral orchiectomy was done on July 31. Serum acid phosphatase on July 27, was 3.35 Gutman units and alkaline 15 King-Armstrong units.

Tissue report of the right testicle. Grossly, there were firm, discrete, yellow nodules scattered through the testicle. Microscopically, (fig. 3) there were anaplastic cells resembling those in portions of the prostatic carcinoma. Cells showed hyperchromic nuclei and scant cytoplasm. The left testicle was normal.

He was discharged from the hospital on Aug. 31, on a maintenance dose of 3 mg. of stilbestrol daily. He was called back for a follow-up examination on Dec. 3, 1951. On rectal palpation, the prostate was firm and nodular. There was a mild constriction of the vesical neck, but no residual urine was found. Stilbestrol dosage was increased to 5 mg. daily.

On Jan. 1, 1952, the patient was rehospitalized because of severe pain in the low back and right hip. Roentgenograms of the chest, lumbosacral spine, and pelvis failed to reveal bone metastases. On Jan. 23, serum acid phosphatase was 5.1 Gutman units and on Jan. 29, it was 6.1. Stilbestrol dosage was increased on Jan. 23, to 25 mg. four times daily orally, and 10 mg. in oil intramuscularly daily. The back and right hip pain had subsided by Feb. 4, when he was discharged on a maintenance dose of stilbestrol.

His last hospitalization began on March 3, because of recurrence of severe low back and right hip pain. Roentgenograms of the chest, lumbosacral spine, pelvis, right femur and right knee still failed to show bone metastases. The serum acid phosphatase was 14 Gutman

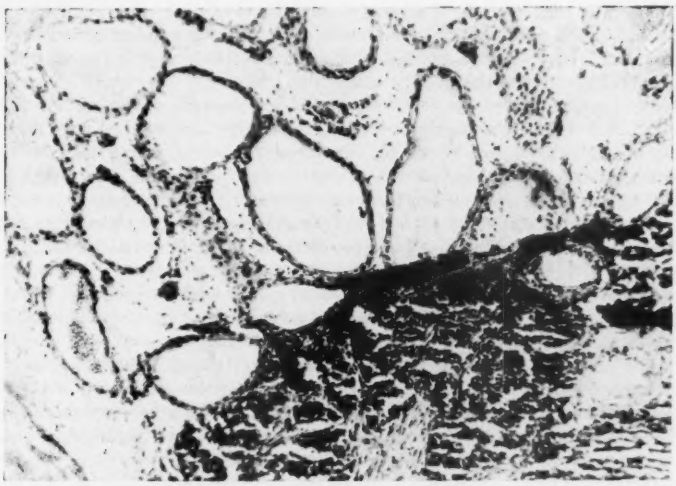

FIG. 3. Testicular metastasis from carcinoma of the prostate in case 2

units on March 25, and 17 on April 22. He gradually became weaker, and expired on July 2, 1952. Autopsy permission was not granted.

Case 3. J. N. Z., a 73 year old white man was seen first on admission to the Lake City Veterans Administration Hospital on Nov. 19, 1952. He complained of a sudden recurrence, on Nov. 16, 1952, of paralysis of both legs, numbness from the waist down, and incontinence of feces and urine.

Five years before the present admission, prostatic carcinoma was found at operation elsewhere. In September 1951, a bilateral orchiectomy had been done during a 4 months' period of paraplegia. He was then also placed on oral estrogenic therapy, and recovered the use of his legs until the sudden onset of the present episode.

He was a well developed and nourished 73 year old white man with paralysis of both lower extremities. The patellar and achilles tendon reflexes were absent, but the Babinski's reflex was present. Skin anesthesia was present up to the nipples. Both testes were absent, and there was an indwelling urethral catheter. The prostate gland was small, flat, smooth and firm.

On admission, the blood count and chest roentgenogram were normal. A serologic test for syphilis was negative. The serum alkaline phosphatase was normal. The serum acid phosphatase was elevated to 5 Gutman units and the sedimentation rate was 14 mm. The blood calcium was 14 mg. per cent and phosphorus 3 mg. per cent. There were osteoblastic metastatic changes in the thoracic (7, 8, 9, 12) and lumbar vertebrae (1, 2), and in the anterior portion of the right fifth rib. These findings were shown on a series of roentgenograms.

Remarkably, the patient had no pain whatsoever. He was treated with stilbestrol 25 mg. daily intramuscularly, and TACE, 12 mg., four times daily orally. By Dec. 11, 1952 the serum acid phosphatase was 2 Gutman units and the sedimentation rate was 12 mm. During his hospitalization, the level of anesthesia receded from the nipple line to the umbilicus, with beginning return of sensation over the thighs anteriorly.

On Jan. 5, 1953, he was transferred, at his own request, to the Dearborn Michigan Veterans Administration Hospital to be near his home. While at that hospital, on January 11, he suddenly developed an episode of dyspnea, cyanosis, and a weak rapid pulse. The cardiac rhythm showed frequent extrasystoles. The blood pressure was 160/80. The left chest showed limitation of motion and absence of breath sounds. He died on Jan. 12, 1953 in spite of cardiorespiratory supportive measures.

An autopsy was done. Grossly it showed consolidation of the lower and middle lobes of the left lung, a small firm prostate, diffuse osteosclerosis of the thoracic vertebral bodies (7, 8, 9, 10), and lumbar vertebral bodies (1, 2), and a cystic change in the central portion of the lower thoracic area of the spinal cord.

Microscopic study showed the following pathologic changes: a thickened pleura with small collections of irregular malignant acini, especially localized around bronchioles, blood vessels and walls of larger bronchi, infiltration of several portal spaces of the liver specimen with malignant acinar cells, nests and cords of malignant cells in the prostate with small, hyperchromatic nuclei, mitoses not abundant, areas of demyelinization of the anterior, lateral, and posterior columns of the thoracic spinal cord, with anterior and posterior horns showing some compression but no evidence of malignancy, and marked thickening and sclerosis of the vertebral bone (fig. 4) with malignant glands seen in the interspaces.

An additional case of paraplegia secondary to spinal cord compression by vertebral metastases also will be presented. This diagnosis was confirmed by clinical findings, roentgenography, and elevated serum acid phosphatase level. An autopsy was not permitted.

Case 4. J. O. R., a 61 year old white man, first was hospitalized at the Lake City Veterans Administration Hospital from June 17 to Sept. 23, 1952. A needle biopsy of the prostate taken on June 23, was positive for carcinoma. Serum acid phosphatase and roentgenograms of the chest, spine and pelvis were negative. On July 28, a radical perineal prostatectomy was done, and the surgical specimen confirmed the diagnosis of carcinoma of the prostate.

He was seen for a follow-up examination on Dec. 16, and hospitalized because of pain in the thoracic and lumbar spine. Roentgenograms of the chest and thoracic and lumbosacral

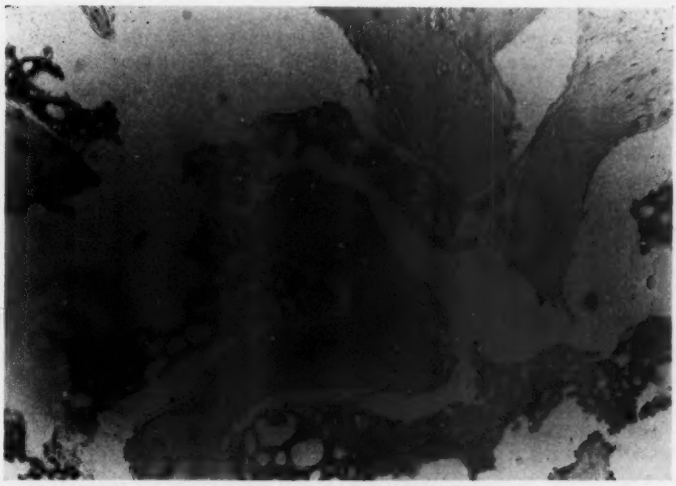

FIG. 4. Sclerosis of the vertebral bone, showing malignant glands in the interspaces in case 3.

spine failed to show any metastases. The serum acid phosphatase level was 3 Gutman units. By Dec. 19, 1952, paresthesias, weakness and pains in both legs became so severe that he was unable to walk. Examination of the lower extremities showed hyperactive reflexes, marked incoordination and diminished skin sensation. On Jan. 5, 1953, the spinal fluid was found to be clear, with increase in globulin, and 290 mg. per cent of total protein.

During the first 5 months of 1953, he was treated with cortisone and estrogens. The leg pains disappeared, but the weakness and incoördination remained.

Spine roentgenograms of April 13, and June 2, and chest roentgenograms of April 29 and June 25, did not show metastases.

Estrogen and cortisone therapy were discontinued during June and July. On July 24, roentgenograms of the thoracic and lumbar spine, pelvis and hips showed extensive demineralization and loss of calcified bone mass. Similar roentgenograms were obtained on September 2, October 27 and December 7. The last films showed a vertical compression of the body of the eighth thoracic vertebra and, biconcavity of several bodies of the thoracic vertebrae. The skull, hands and feet bones showed considerable decalcification on November 17.

Many serum acid phosphatase determinations were made during 1953 and these varied from 0.9 to 4 Gutman units. Gastrointestinal barium study, cholecystogram and intravenous pyelography were made during the first week in July 1953; they all were negative.

Estrogen and cortisone were given again until the last week of December 1953, when testosterone was substituted until the end of January 1954. This was done to determine the effect on the bone lesions and the serum acid phosphatase level. The serum acid phosphatase level rose from 1 to 11 Gutman units, and the alkaline phosphatase from 32.5 to 75 King-Armstrong units. Hypercalcemia (14 mg. per cent) and hyperphosphatemia (6 mg. per cent) also occurred. Subjectively, the patient developed severe nausea and vomiting, with fever of two weeks duration and prostration. These symptoms probably were on the bases of the hypercalcemia, which was further complicated by some degree of adrenocortical insufficiency. The patient improved in a few days, with the administration of cortisone, intravenous glucose, intravenous electrolytes and antibiotics.

Estrogenic therapy was resumed on Feb. 25, 1954 and continued in combination with cortisone until the patient's death. On May 28, 1954, roentgenograms of the entire spine and pelvis showed disseminated osteoblastic lesions for the first time.

During most of his hospitalization, he was up and about in a wheel-chair. He did not exhibit the typical paraplegic changes in his legs, such as loss of cutaneous sensation, advanced muscular atrophy, and complete paralysis, until the last four weeks of his illness. He deteriorated rapidly during this time complaining of severe pains in the thoracic spine and legs.

Regardless of the lack of corroboration by an autopsy, the author believes that there is sufficient evidence to warrant the diagnosis of paraplegia, secondary to spinal cord pressure by vertebral metastases of prostatic carcinoma. The unusual early bone changes of loss of calcified bone may have been due to a combination of factors, including the cortisone therapy, atrophy of disuse, an estrogen–androgen imbalance, and bone metastases of the prostatic malignancy. When the bone lesions finally demonstrated increased bone density, the osteoblastic activity of the prostatic metastases to bone became apparent.

SUMMARY

Case reports of three rare metastatic sites of carcinoma of the prostate are recorded. The metastases were to the corpora of the penis, testicle and to the vertebrae, the last causing compression of the spinal cord.

Additional Note:

Since this article was submitted for publication, Hamm and Weinberg* reported two cases of metastasis of prostatic carcinoma to the penis, and Espinosa and associates** reported one.

This increases the prior total number of recorded cases from 17 to 20.

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SURGICAL TECHNIC

DISPLACED FRACTURES OF THE LOWER FOREARM IN CHILDREN: USE OF THE FLEXION-REVERSE FLEXION TECHNIC OF DE SOUZA RAMOS

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A frequently encountered injury in younger children is a transverse fracture of the radius and ulna 1 inch, more or less, above the distal epiphysis. A fair number of these have a complete dorsal displacement of one or both lower fragments with overriding and shortening. This type of deformity presents some difficulty in reduction and is the subject of this paper.

The correction of shortening due to overriding of fragments may, as a rule, be obtained by direct longitudinal traction and counter-traction. The force required to restore length by this means may be considerable. If, however, end to end apposition first is obtained by deliberate angulation, then (assuming the ends to be engaged) alignment and length are easily restored and maintained. This principle has been clearly brought out by De Souza Ramos[1] and it is our purpose here merely to cite another of its many possible applications.

In displaced lower forearm fractures in children, strong longitudinal traction may not permit restoration of satisfactory apposition of the fragments of the radius and ulna. This particularly is true if the displacement is of several days' duration, or when swelling is severe, as is the case after repeated forceful manipulations. It then becomes eminently desirable to quickly and gently reduce the displacement (fig. 1).

The difficulty experienced by us in the reduction of these fractures led to the employment of a method of reduction which employed the flexion-reverse flexion principle.

TECHNIC

Good relaxation under general anesthesia, such as is provided by drop ether, is very helpful. Strong traction then is applied to the hand, and counter traction to the flexed elbow to stretch the soft parts as a preliminary to the reduction maneuver. The thumbs are placed on the dorsum of the forearm, at the point of palpable deformity, and the hand and distal fragments are firmly grasped by both hands of the operator (fig. 2). The deformity then is increased by dorsiflexion of the lower fragments of radius and ulna (fig. 2 B). Maintaining this increased deformity for a moment, the operator's thumbs press directly distalward against the dorsum of the lower fragments, and this pressure is prolonged until the shortening is judged to be overcome (fig. 2 C). The alignment of upper and

FIG. 1. Displaced distal forearm fracture, showing displacement

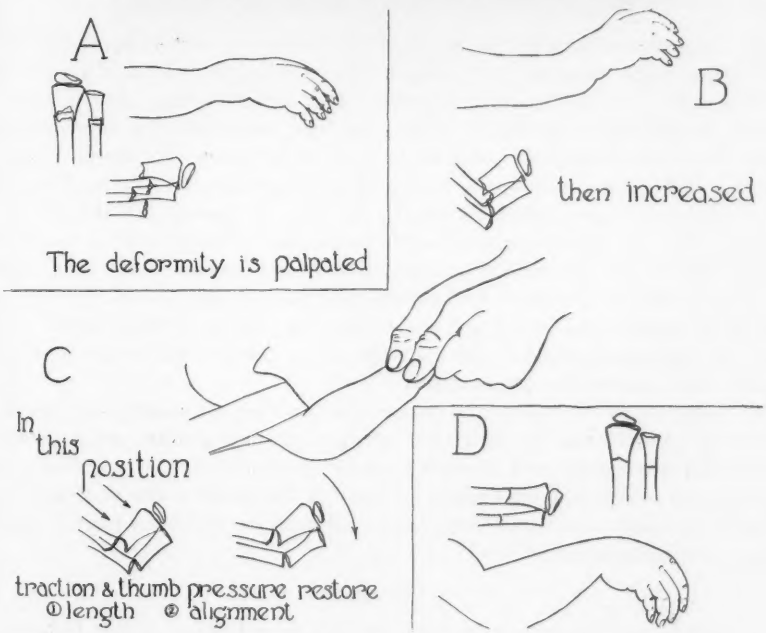

FIG. 2

FIG. 3. X Ray film showing reduction of the fractures of the radius and ulna by the technique described.

lower fragments, meanwhile, is such that the fragments meet at approximately a 90 degree angle. Once length is judged to have been restored, the alignment is immediately corrected by *downward* pressure on the lower fragments, or *reverse flexion*. If sufficient longitudinal pressure has been made while the fragments remain flexed, the straightening maneuver completely restores alignment and apposition of the fragments, usually to an *anatomic* degree (fig. 3). The hand should be allowed to fall into moderate flexion by the action of gravity, in which position the reduction is very stable and roentgenograms may be taken without danger of displacement. The cast should incorporate pads over prominences—other light padding to protect the skin—and should be split from end to end. It must, of course, immobilize the elbow, and should hold the wrist moderately flexed. Distally the cast must extend to the knuckles on the dorsum, and to the proximal crease of the palm on the volar surface.

Although clinical union may be present in a few weeks, the solid bony healing necessary to withstand the rigorous demands of playing children is not present for at least two months, and frequently not for three months. The cast should not be removed until roentgenographic evidence of the fracture line is absent. To remove the cast sooner will invite many refractures, which frequently occur after a surprising length of time.

RESULTS

Uniformly satisfactory results have resulted from the use of this technic of reduction. Occasionally, the maneuver must be repeated to correct all displace-

ment. In arms not previously manipulated or badly swollen, something approaching anatomic reduction may be expected.

SUMMARY

The use of the method of reduction by flexion and reversed flexion, first clearly stated by De Souza Ramos, is suggested in displaced fractures of the lower radius and ulna in children. Using his principle, reductions of such fractures are obtained with remarkable facility and without the exertion of force as is required in the use of direct longitudinal traction. Immobilization in these fractures should include the elbow and hold the wrist in flexion for a minimum period of two months for optimum healing.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS WITH SUCCESSFUL REPAIR BY SUBDIAPHRAGMATIC APPROACH

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HISTORY

Spontaneous rupture of the esophagus should be distinguished from perforation, the latter term being used where the rupture follows disease of the esophagus or is incident to instrumentation or a perforating missile.[7] This condition was first described by Boerhaave in 1724.[10] The first operative cure was reported by Barrett in 1947[5] following repair through a thoracotomy incision. Collis[11] first attempted repair of a ruptured esophagus in 1944, but the patient did not survive. Various types of operative treatment have been tried in the past 20 years, mostly consisting of drainage through the superior mediastinum or through the posterior mediastinum with or without suture of the defect, and more recently, approach through the intercostal incision with suture of the esophagus and drainage of the mediastinum.

SIGNS AND SYMPTOMS

The characteristic symptom of spontaneous rupture of the esophagus is pain. The pain is substernal or sometimes is felt in the epigastrium. It is referred to the left side of the back. It is intense in character, being so severe that repeated hypodermic injections of morphine often fail to relieve it. It characteristically comes on after a severe vomiting spell, especially following over-eating or over-indulgence of alcohol. Curiously enough, the vomiting usually stops after the esophagus ruptures. The patient exhibits severe apprehension and cyanosis rapidly develops. Crepitus often is felt in the neck and emphysema of the mediastinum and other adjacent tissues can be seen in the roentgenogram. Abdominal rigidity is a very characteristic sign, together with marked tenderness. The patient rapidly goes into a state of severe shock; dyspnea develops and he usually dies within a few hours if operative intervention is not forthcoming. Roentgenographic examination of the chest often shows fluid or air in the pleural cavity;[11] however, none has been described in the abdomen.[8] Roentgenographic examination of the esophagus, using lipiodol, not barium, will show the ruptured esophagus without difficulty.

DIFFERENTIAL DIAGNOSIS

Some of the conditions to be thought of in the differential diagnosis are:

1. Coronary occlusion.
2. Dissecting aneurysm of the aorta.
3. Perforated peptic ulcer.
4. Spontaneous pneumothorax.
5. Spontaneous mediastinal emphysema.

6. Pulmonary embolism.
7. Acute pancreatitis.
8. Incarcerated diaphragmatic hernia.
9. Rarely, bronchopleural fistula.

PATHOLOGY

The rent in the esophagus has been found always to be on the left side just above the diaphragm.[11] Various reasons have been advanced for this peculiarly constant finding. Angulation in the esophagus at that point, with natural thinning of the left side of the esophagus, together with the fact that the right side is supported to some extent by the aorta, retrocardiac fat and the vertebral bodies have been suggested to explain this peculiarity.[11] The tear in the esophagus almost universally is longitudinal.[3] In most of the cases reported in the literature, the cause of the rent in the esophagus is increased intraabdominal pressure, as may be seen in severe vomiting, retching, straining at childbirth, straining at defecation, weight-lifting, recovery from anesthesia and severe blows on the abdomen. Much experimental work has been done on fresh cadavers and it can be shown that the esophagus will rupture with hydrostatic pressures of from 2 to 6 pounds and that the rupture always will be just above the diaphragm on the left side.[3] Eighty-four per cent of the spontaneous ruptures of the esophagus have been reported in males; the youngest was five years and the average about 40 to 60 years of age.[6]

TREATMENT

The only treatment which has produced satisfactory results has been operative. Fogett[11] in 1946 repaired and drained a ruptured esophagus through the abdomen, but the patient died immediately of mediasinitis. The best results are seen if operation can be done within the first 15 hours.[9] One patient was reported to have recovered after laparotomy and drainage with the spontaneous closure of a fistula which developed; however, all of the other recoveries in the literature have followed thoracotomy and closure of the esophageal rupture with drainage of the chest. Wangensteen suction is employed in all cases, together with the use of large doses of antibiotics.

COMPLICATIONS

Postoperative complications have been empyema and suppurative mediastinitis.[2] It is believed that the pleura often may remain intact for as long as 15 hours, especially if the stomach happens to be empty at the time of the injury.[6]

CASE REPORT

A 64 year old retired farmer, who previously had had good health was seen in the hospital emergency room about midnight, giving a history of having taken 2 teaspoonfuls of Saraka at about 10:05 p.m. The Saraka seemed to lodge in the esophagus and he felt considerable discomfort following the swallowing of it. Thinking that more fluid was to be required to wash the Saraka into the stomach, he drank a Dr. Pepper. The Dr. Pepper failed to relieve the pain. It gradually increased and when he arrived at the hospital, it was quite severe.

It was substernal in location and also was felt in the epigastrium and radiated into the left side of the back. It was definitely spasmodic in nature. He would be relatively free of pain and then would *double up*, according to the observing physician. The pain was unrelieved by 3 hypodermic injections of one-fourth grain of morphine. He was admitted to the hospital and diagnostic study was begun.

Two O'clock a.m.: His blood pressure was 110/70, and the pulse 72, with good volume. The electrocardiogram was normal. A roentgenogram of the urinary tract showed nothing diagnostic. A lateral decubitus film of the abdomen showed no evidence of free air in the peritoneal cavity. A chest roentgenogram was reported as not being helpful, but later examination showed some evidence of a little fluid in the left lower pleural cavity.

Three O'clock a.m.: He was given an enema, which was retained and could not be expelled. It gave him no relief. Abdominal rigidity was developing and the pain in his left chest and subdiaphragmatic and substernal areas was becoming related to breathing, being worse on deep inspiration. A catheterized specimen of urine was normal.

Four O'clock a.m.: The patient was becoming nauseated. His blood pressure was 130/80, and pulse of good volume. Nitroglycerin failed to relieve the pain. Morphine and atropine were repeated. At this time, surgical consultation resulted in the following differential diagnoses:

1. Acute pancreatitis.
2. Ruptured peptic ulcer.
3. Dissecting aneurysm of the aorta.
4. Coronary occlusion.
5. Rupture of the esophagus.

Five-thirty O'clock a.m.: A medical consultant was called and on his examination at 6:30 a.m., emphysema was first noted in the left side of the neck and a roentgenologic study of the esophagus was advised. The esophageal roentgenograms, using lipiodol, showed evidence of a ruptured esophagus, just above the cardioesophageal opening. At that time his blood pressure was 114/60, and his pulse was becoming faster and of poor volume. He rapidly became cyanotic. He was operated upon immediately. The area was approached through a wide transverse upper abdominal incision, transecting both recti muscles. On opening the peritoneal cavity, considerable colored fluid was encountered with flecks of fibrin, much as is seen in a case of ruptured peptic ulcer. No evidence of an ulcer was found, but it was noted that there was subserous emphysema involving the upper half of the stomach. The left leaf of the triangular ligament of the liver was divided and the left lobe of the liver was retracted to expose the cardiac end of the stomach. The reflection of the peritoneum at the cardiodiaphragmatic junction was opened and the esophagus was brought down through the hiatus for inspection. A rent in the esophagus about 4 cm. long was immediately detected on the left side beginning about 1 cm. above the cardiac opening. This was immediately closed with cat-gut and a large penrose drain was placed through the hiatus well up into the mediastinum and brought out through a stab wound in the epigastrium. The abdomen then was closed and the patient was returned to his room in an improved condition.

He was given oxygen constantly. Gastric suction was continued and large doses of antibiotics were administered. His pulse ranged to 148 the first postoperative day. The second postoperative day, he appeared better. His pulse was 120. His color was good. His temperature rose to 102.6 F. rectally. On the third postoperative day, further improvement was noted. His abdomen was soft and his maximum temperature was 101.8 F. rectally. On the fourth postoperative day, his pulse suddenly rose to 160 and an electrocardiogram disclosed a supraventricular tachycardia. This responded dramatically to the administration of quinidine intravenously. On the fifth postoperative day, a pericardial friction rub could be noted and there was some evidence of a possible pericardial effusion. The Levine tube was removed on the sixth postoperative day and the penrose drain shortened. A profuse purulent drainage had been present since the operation, but now it was becoming less. The Levine tube was removed on the tenth postoperative day and the patient, who had been fed previ-

ously through the tube, now was allowed a soft diet. On the twelfth postoperative day, some roentgenographic evidence of fluid in the left pleural cavity resulted in the aspiration of 100 cc. of serosanguineous fluid which showed no organisms. At this time he was afebrile and was dismissed a few days later, apparently recovered.

CONCLUSIONS

So far as is known, this is the first case of spontaneous rupture of the esophagus to have been reported due to one of the vegetable hydrogels (Saraka). Also, so far as is known, this is the first patient to have recovered following subdiaphragmatic repair of the esophagus and drainage of the mediastinum.

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TREATMENT OF INFECTED URACHAL CYST BY PRIMARY EXCISION: A REPORT OF TWO CASES*

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Urachal cysts are quite rare, and as clinical entities, they are extremely rare since they are usually entirely asymptomatic unless they become infected. Treatment of the infected urachal cyst has been the subject of several excellent articles in the recent literature. Most authors, among them Sibley,[2] Tauber and Bloom,[4] have advocated simple incision and drainage, either with or without marsupialization.

The primary purpose of this paper is to report 2 cases of infected urachal cyst, 1 in an adult and 1 in an infant, both successfully treated by primary excision. For a review of the embryology and anatomy of the urachus, the reader is referred to Brodie's[1] excellent paper.

CASE REPORTS

Case 1. The patient is a 29 year old white man who always has enjoyed excellent health. He first was seen by one of us (J.C.R.) on Nov. 20, 1954. with a chief complaint of pain and tenderness in and immediately below the umbilicus since Nov. 15, 1954. At the time of the original examination he stated that a milky discharge had begun the night before. Physical examination showed some reddening of the skin of the umbilicus, this skin being covered with a thin milky discharge. There was tenderness directly over and immediately below the umbilicus. The patient was told at that time that he might have a urachal cyst. He was started on treatment with hot soaks and antibiotics and seemed to be improving when he was seen again three days later. On November 25, the pain increased noticeably in severity and the discharge increased in quantity. He was admitted to the Presbyterian Hospital Center.

System review was entirely negative except for the present illness. He stated that he had never had umbilical drainage before. He had no genitourinary complaints. The physical examination was within normal limits with the exception of the abdominal wall findings. The umbilicus, centrally, was reddened and moist with a milky, nonodorus discharge. There was exquisite tenderness on either side of the umbilicus and inferior to it. There was no tenderness above. A tender mass was felt immediately below the umbilicus, extending several centimeters in the direction of the symphysis pubis. No abdominal masses were palpated elsewhere. The admission diagnosis was infected urachal cyst, and the alternate possibility of a communicating Meckel's diverticulum.

Admission blood count was normal. The urinalysis was normal. Examination of the abdomen by roentgenogram in the anteroposterior and lateral views showed no demonstrable soft tissue mass. Cultures of the discharge did not show growth.

The patient was placed on bed rest with warm saline soaks to the umbilicus. He was given antibiotics. The first night in the hospital there was much more drainage and the following morning the tenderness, especially to the left of the umbilicus, had increased noticeably. In addition, rebound tenderness had developed. It was thought that attempts at injecting lipiodol through the umbilical opening might prove to be a hazardous procedure. Technically this would have been extremely difficult for the opening could not be visualized be-

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cause of the swelling of the cutaneous tissues. Because of the possibility that the discharge was caused by a communicating Meckel's diverticulum, it was considered unwise to attempt incision and drainage. Preparations for surgery were made, and the patient was taken to the operating room on the afternoon of November 26, approximately 16 hours after admission.

Operative procedure. Under spinal anesthesia the abdomen was opened through a midline incision approximately 1 inch beneath the umbilicus. The reason for entering the abdominal cavity was to ascertain whether or not the mass was an urachal cyst or possibly a Meckel's diverticulum communicating through the umbilicus. Upon opening the abdominal cavity free fluid was not found. Direct visualization of the posterior aspect of the umbilical region showed a cystic mass measuring about 4 cm. longitudinally and 2 cm. laterally, and approximately 2 cm. thick. The overlying peritoneum was extremely reddened and was covered by exudate. The urachus, which was a small fibrous cord, was found to be in continuity with the inferior border of the mass. During operation, at no time was there any discharge from the umbilicus.

After determining that the mass was an infected urachal cyst, the umbilicus was held closed with Allis forceps while it was excised. The entire operative field was adequately walled off both before and during the excision of the umbilicus. Following its removal, all sponges were discarded as were all instruments used in the excision. The tissue excised included the umbilicus with the umbilical ring, and the infected urachal cyst (fig. 1) with an adequate margin of the peritoneum on either side, the urachus being severed inferiorly, and ligated. Following this the abdominal wall was closed in layers and the patient was returned to his room in good condition.

FIG. 1: Photograph of the surgical specimen (case 1). The umbilicus and the cyst have been opened. Note that the inflammatory reaction includes the peritoneum.

Postoperatively the patient made an uneventful recovery. The microscopic examination showed that the lining of the cyst had been largely destroyed, and that the cyst cavity was filled with purulent exudate. The necrotizing inflammatory process extended to within 2 mm. of the peritoneal surface. There was no evidence of specific inflammatory reaction. Pathologic diagnosis: "Urachal cyst abscess with incipient peritoneal perforation." The patient was discharged from the hospital on November 30, his fourth postoperative day. Sutures were removed on the seventh postoperative day. The wound remained clean and the resulting scar is well healed. There is no weakness of the abdominal wall. The patient is, at present, (May 1, 1955) back at work and asymptomatic.

Case 2. The patient was a 3 months old male infant, who first was seen by one of us (J. C. R.) in the office on Jan. 27, 1955. The occasion for the examination was a routine monthly infant examination. At the time the baby was asymptomatic. At that examination a small umbilical hernia was reduced and a strip of tape was placed across the skin of the umbilical region. On February 9, the mother phoned to report that the baby had been exceedingly irritable the preceding four days and that she had just removed the tape from the umbilical region and noticed extreme redness of the umbilicus with a clear thin fluid covering the entire region. She was advised to bring the child to the hospital for further examination. He was seen in the emergency room and admitted to the Presbyterian Hospital Center on February 9, at 4:30 p.m.

The physical examination essentially was within normal limits with exception of the abdominal findings. The abdomen was somewhat protuberant. Numerous fecal particles were palpated in the region of the descending colon. The umbilicus was protruding and its surface was reddened. Near the right lower quadrant of the protruding navel was a small orifice from which a milky type of fluid could be expressed. A soft tumor mass (fig. 2) was

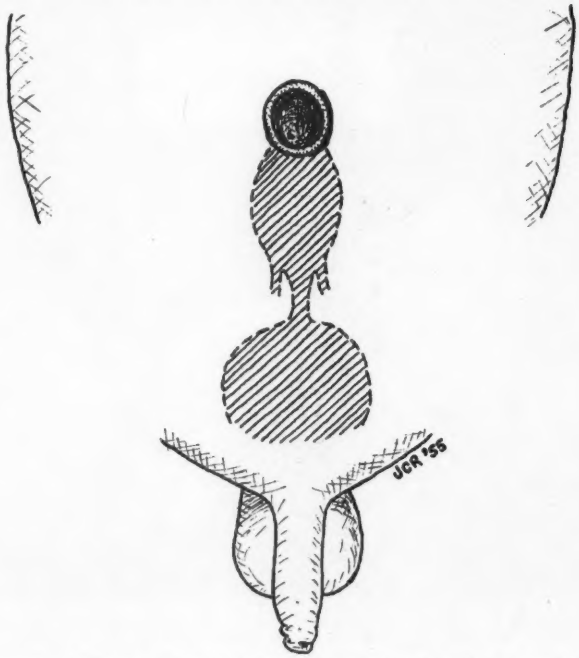

FIG. 2: Schematic drawing of case 2 showing the relationship of the urachal cyst to the umbilicus and to the bladder. The lateral umbilical folds are seen on either side of the urachus.

palpated inferior to the navel for approximately 3 cm. and was approximately 2 cm. across. Compression of this mass produced the escape of milky fluid from the orifice. The mass apparently was within the abdominal wall. An umbilical hernia was not present. The admission diagnosis was "infected urachal cyst."

The admission blood count was normal except for a moderate leukocytosis. The urine was normal. Cultures of the discharge from the umbilicus yielded staphylococcus albus. Roentgenographic examination of the abdomen in the anteroposterior and lateral views revealed a soft tissue mass extending from the umbilicus downward toward the bladder, within the anterior abdominal wall.

The baby was started on antibiotic therapy. The lower bowel was emptied with several cleansing enemas. He was operated upon on February 10.

Open drop ether anesthesia was used. The skin edges on each side of the umbilicus were grasped and held together with Allis forceps to prevent contamination during the surgical procedure. The abdomen was opened through a midline incision extending from just below the umbilicus to approximately 1 cm. above the symphysis pubis. The dissecton was carried down through the rectus fascia, and the bellies of the recti were separated laterally revealing a cystic mass in the midline. Attempts to separate the mass from the underlying peritoneum were unsuccessful. The mass was adherent to the peritoneum because of the inflammatory reaction. Accordingly, the peritoneal cavity was entered below the mass and to the right of the thickened urachus in order to facilitate excision.

The mass, which extended from the posterior aspect of the umbilicus (fig. 3) and extended inferiorly toward the symphysis in the midline, measured approximately 4 cm. in length and 2.5 cm. in width. At its inferior end the cord-like enlarged urachus continued on

FIG. 3: Case 2. Schematic drawing showing a lateral view of the urachal cyst

down to the bladder and was found not to be patent at that point. It was doubly ligated at its bladder attachment. The bladder appeared normal. An elliptical incision was developed in the peritoneum on each side of the cystic mass and extended from the umbilicus on its posterior aspect down to the severed urachus. The incision included the severing of the lateral umbilical folds, each of which was ligated. Elliptical incisions were developed on each side of the umbilicus (fig. 4) down through the subcutaneous tissues and the fascia of the recti, thus facilitating the removal of the umbilicus along the urachal cyst, and a portion of the urachus. After excision of the umbilicus and the urachal cyst, the abdomen was closed in layers. The patient tolerated the procedure very well.

The microscopic report showed the internal lining of the cyst to have been destroyed, and to have been replaced by highly vascular granulation tissue which was diffusely infiltered by large numbers of neutrophils and small numbers of lymphocytes and mononuclear phagocytes. There were no specific inflammatory changes. The microscopic diagnosis: "Urachal cyst with acute and nonspecific inflammatory reaction." Subsequent to operation, the baby made an uneventful recovery. The antibiotic therapy was continued and on February 14, the fourth postoperative day, he was discharged from the hospital. Sutures were removed on the seventh postoperative day. He has remained asymptomatic (May 1, 1955). The incision is well healed and there is no weakness of the abdominal wall.

COMMENT

The usual treatment of infected urachal cysts has, heretofore, consisted of incision and drainage, either alone or combined with marsupialization. Although

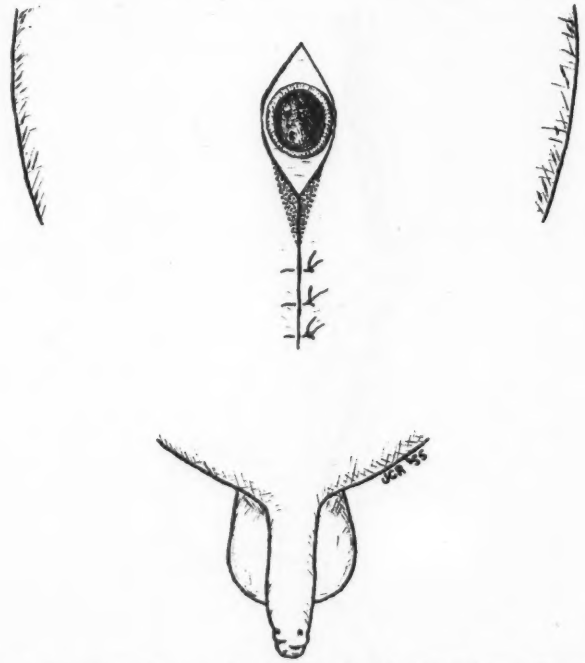

FIG. 4: Case 2. Schematic drawing showing location of skin incisions. Shaded areas represent skin excised to facilitate straight line closure. The same technic was used in both cases.

effective, this form of treatment carries with it a great potential risk, namely perforation of the cyst wall into the abdominal cavity, during or subsequent to operation. Furthermore, the end result of such treatment, we believe, falls short of the real surgical goal, the removal of the diseased tissue. The authors suggest that infected urachal cysts be treated by primary excision. Antibiotics should, of course, be used preoperatively and postoperatively.

SUMMARY

Two cases of infected urachal cyst are presented. Both patients were successfully treated by primary excision, made possible by the use of antibiotics, preoperatively and postoperatively. The surgical approach is described.

The first case is unique in that the patient was entirely asymptomatic until the age of 29 years. Sterling and Goldsmith[3] have stated that it is uncommon to see adults with inflammation in the urachal tract who had no symptoms in infancy. Attempt to inject lipiodol into the cyst preoperatively was considered impractical, and, in retrospect, such an attempt might well have been followed by generalized peritonitis. Incision and drainage, we believe, would have been extremely hazardous because of the degree of inflammation of the cyst wall and the underlying peritoneum.

The second case is unique in that there were no symptoms until nine days after a small umbilical hernia was taped.

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EDITORIAL

ACUTE CHOLECYSTITIS

The label *acute cholecystitis* is attached to a symptom complex in which there is characteristic pain in the upper abdomen accompanied by varying other signs and symptoms. The finality of this diagnostic term leads to impressions that the disease is an entity; that criteria for the diagnosis are definite and that there is understanding of cause. These impressions are false.

The established facts are: in patients whose diagnosis is labeled thus there frequently are, but not always, stones or inflammation in the gallbladder; and empirically it has been determined that removal of the gallbladder frequently will, but not always, prevent further attacks.

The gallbladder is an appendage of a complicated and important glandular and duct system derived from the gastrointestinal tract. The whole system comprises the pancreas and liver, hepatic bile ducts and pancreatic ducts, duodenal wall, the gallbladder and a specialized section of circular muscle fibers—the sphincter of Oddi. The gallbladder fills passively and the sphincter of Oddi creates the ductal pressures to which the gallbladder is responsive. In animals which have no gallbladder, e.g. rat or horse, the sphincter of Oddi also is missing. Cholesterol stone formation undoubtedly is associated with disordered reabsorption of components of bile in the gallbladder. This concentration of bile by gallbladder mucosa is altered by intraductal pressure changes. The process is dynamic rather than static. That is, stone formation may be accompanied by hypertonic activity rather than atony or stasis. Certainly the basic disturbance is not limited to the gallbladder itself.

Secretion by the liver and pancreas provides the pressure head of the closed system and the sphincter of Oddi at the common outlet is the peripheral resistance. Flow of juice and lateral pressures in the ducts are resultants of these forces. Both in turn are influenced by gastric secretion reaching the duodenum, duodenal and jejunal secretion of intestinal hormones, pyloric sphincter function and autonomic nervous stimuli.

Acute complications arising in the course of disease of this system may be:

(1) Gallstone colic.

(2) Reflux of activated pancreatic juice up the common bile duct, fat necrosis and chemical proteolysis of the gallbladder.

(3) Obstruction of the cystic duct by stone, secretion of mucus into the gallbladder.

(4) Distention or inflammation of bile ducts without gallbladder disease.

(5) Acute pancreatitis or hepatitis.

(6) Extrusion of a stone from the gallbladder into a nearby hollow viscus.

If bacterial inflammation supervenes it is secondary. There is no evidence that bacteria initiate the acute attacks and there is no analogy between so-called acute cholecystitis and ruptured intestinal tract. Leakage of bile or bile-stained fluid through the gallbladder wall is not infrequent and is almost always confined to

the immediate area by adherent omentum. Necrosis of the gallbladder wall is a peculiar spotty pattern and leakage of bile similarly is walled-off quickly and efficiently. The terms *gangrene* and *perforation* used under such conditions are foreboding beyond the actual circumstances. During operation upon an acutely inflamed gallbladder, dissection of adherent omentum from the wall may disclose leakage of bile. This does not mean that the patient would have died if the operation had not been done.

It has been demonstrated clearly that operative treatment of the acutely inflamed gallbladder can be carried out with relative safety and low mortality under proper auspices. It also has been clearly demonstrated that, with proper nonoperative measures, the acute attacks which occur in the course of chronic disease of the system will rapidly subside. In the latter case operation is delayed and is performed electively. The large question is not what *can* be done, but what *should* be done.

Factors other than mortality and morbidity statistics from good centers should be considered in such decisions. Some of these are: the appallingly large incidence of common duct injuries which the literature in this country reveals; the fact that in one studied series* of 116 patients diagnosed as acute cholecystitis 19 were shown by further study to have acute pancreatitis, a condition in which operation is not indicated; the considerable number of patients who must undergo further operation after initial attack during the acute phase.

It is misleading to describe the *edema phase* of acute inflammation as an aid in dissection of the gallbladder. Such edema about the common duct and retroperitoneal vessels is a hazard.

The rationale of nonoperative treatment of acute complications is to reverse intraductal pressure and flow disorganization by:

(1) Continuous suction through a nasal gastric tube to prevent access of hydrochloric acid to the duodenum.

(2) Sedation for pain, preferably with meperidine. Morphine causes smooth muscle spasm which is objectionable and might increase the difficulties. For anxiety and apprehension-intramuscular barbiturate is of benefit.

(3) Anticholinergic drugs to relax spasm of the sphincter of Oddi.

(4) Antibiotics to control infection. The hazardous effect of free bile in the peritoneal cavity is minimized by penicillin.

In most instances pain subsides in a few hours, and in three to five days all symptoms have receded. In occasional instances of abscess formation drainage may be necessary, and in others decompression of the gallbladder by cholecystostomy may be deemed advisable. In the series of 119 patients quoted above such intervention was carried out in three.

Many patients with these biliary-pancreatic emergencies are in an age group in which generalized diseases of other systems are present. Delayed operation permits careful evaluation of such accompanying disorders and ample time to provide optimum preparation.

* Reference—Doubilet, Reed and Mulholland. JAMA August, 1954, Vol. 155, pp. 1570-1573.

Objection to deferment of operation might be that the patient will refuse operation when all symptoms have subsided and thus be subject to later, and perhaps more severe, attacks. This is a facet of the art of surgical care and involves a responsibility which cannot be evaded by precipitous action. Further objection may be that the physician or surgeon might undertake to treat the acutely ill patient at home, or regard the nonoperative measures in the hospital as simply waiting. Both are fallacious. The steps to be taken in the reversal of the process are as definite as operation; require thought, judgment and active care, and can be done properly only in a hospital.

The gallbladder is a prominent, accessible, expendable organ which is easily removable by operation. There are excellent roentgenologic methods of displaying it in the intact human being. It is natural that attention is focused on this prominent structure, particularly as stones are such a common accompaniment of disorders of the complex system of which it is a part. The basic dysfunctions in flow, pressures and smooth muscle tone are intangible and difficult to measure in minute to minute changes. It also is natural to attempt to standardize treatment on the basis of a sound major premise. The diagnosis *acute cholecystitis* is not such a foundation. Too many forces are at work to reduce to a simple equation. In any case, aggressive measures short of operation are effective in what is called acute cholecystitis and provide for a period in which to think about the problem.

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BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

Surgical Forum: Proceedings of the Forum Sessions, Fortieth Clinical Congress of the American College of Surgeons, Atlantic City, New Jersey, November, 1954. W. B. Saunders Company, Philadelphia & London, 1955. Price $10.00.

This volume represents a compilation of 154 papers by 373 contributors under 10 chapter headings with 7 introductions. About one-third of the papers depict research in the cardiovascular field; one-fifth involve research on the gastrointestinal tract, liver and pancreas; and 40 per cent reflect interest in surgical nutrition, body fluids, metabolism, steroids, burns, shock and wound healing. Several papers on cancer and anesthesiology comprise the remainder.

Concise summaries at the end of each paper increase its readability. In general, it reflects the interests of various surgical departments throughout the country and identifies and gives means of expression chiefly to the younger men who are engaged in this work. The outstanding feature of this volume is the addition of the introductory remarks, wherein are summarized, by men pre-eminent in their respective fields, what appear to be the most significant and original contributions. Because this book is an advance publication of work most of which will subsequently be published in detail in the journals, it should be of particular value to those individuals and groups engaged in surgical research.

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BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

Ciba Foundation Colloquia on Endocrinology. Volume VIII. The Human Adrenal Cortex. Editors for the Ciba Foundation: G. E. W. WOLSTENHOLME, O.B.E., M.A., M.B., B.Ch. and MARGARET P. CAMERON, M.A., A.B.L.S. Assisted by Joan Etherington. With 227 Illustrations. Little, Brown and Company, Boston, 1955. Price $10.00.

Handbooks for the General Practitioner. Minor Surgery. By JOHN E. SUTTON, M.D., F.A.C.S., Assistant Clinical Professor of Surgery, Cornell University, Medical College. Diplomate American Board of Surgery with over one hundred illustrations. New York, Landsberger Medical Books, Inc. Distributed solely by The Blakiston Division of the McGraw-Hill Book Co. with the exception of Continental Europe. Price $7.00.

The Principles and Practice of Surgical Nursing. By D. F. ELLISON NASH, F.R.C.S., Assistant Surgeon, St. Bartholomew's Hospital, London; Senior Surgeon, Children's Hospital, Sydenham; Consultant Surgeon, Chailey Heritage Hospital; Lecturer in Surgery, Demonstrator of Practical Surgery, St. Bartholomew's Hospital Medical College; formerly Hunterian Professor, Arris and Gale Lecturer, Royal College of Surgeons; Examiner in Surgery, Queen's University, Belfast, and in the Faculty of Dental Surgery, Royal College of Surgeons. London, Edward Arnold (Publishers), Ltd.; Baltimore, The Williams & Wilkins Co. 1955. Price $7.00.

Lectures on the Scientific Basis of Medicine, Volume III, 1953–54. British Postgraduate Medical Federation, University of London. University of London, Athlone Press, 1955. Price $6.00.